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DATE: Wednesday, March 10, 2004

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☐	L11	l9 and (Cgamma3 or C gamma 3)	0
☐	L10	l9 and (C delta or Cdelta)	1
☐	L9	L7 near5 l1	15
☐	L8	L7 and l1	339
☐	L7	L6 or yac or bac	12086
☐	L6	artificial chromosome$	5928
☐	L5	Cdelta and Cgamma3	1
☐	L4	l1 and (l2 or L3)	1
☐	L3	C delta and C gamma 3	1
☐	L2	C delta near3 C gamma 3	1
☐	L1	IgH	1051

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 NEWS 4 DEC 08 INPADOC: Legal Status data reloaded
 NEWS 5 SEP 29 DISSABS now available on STN
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 NEWS 7 OCT 21 BIOSIS file reloaded and enhanced
 NEWS 8 OCT 28 BIOSIS file segment of TOXCENTER reloaded and enhanced
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 NEWS 10 DEC 08 CABA reloaded with left truncation
 NEWS 11 DEC 08 IMS file names changed
 NEWS 12 DEC 09 Experimental property data collected by CAS now available
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 NEWS 13 DEC 09 STN Entry Date available for display in REGISTRY and
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        databases
 NEWS 18 DEC 22 IFIPAT/IFIUDB/IFICDB reloaded with new data and search
fields
 NEWS 19 DEC 22 ABI-INFORM now available on STN
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        and searchable
 NEWS 21 JAN 27 A new search aid, the Company Name Thesaurus, available in
        CA/CAplus
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        changes
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 NEWS 24 MAR 03 MEDLINE file segment of TOXCENTER reloaded
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                                    ENTRY  SESSION
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=> s artificial chromosom?
L1      11819 ARTIFICIAL CHROMOSOM?

=> s IgH
L2      7110 IGH

=> s C delta or Cdelta
L3      6449 C DELTA OR CDELTA

=> s Cgamma3 or C gamma 3
L4      424 CGAMMA3 OR C GAMMA 3

=> s l1 and l2
L5      67 L1 AND L2

=> s l1 or BAC or YAC
L6      25475 L1 OR BAC OR YAC

=> s l6 and l2
L7      116 L6 AND L2

=> s l7 and l3 and l4
L8      2 L7 AND L3 AND L4

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9      2 DUP REM L8 (0 DUPLICATES REMOVED)

=> d bib abs 1-
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L9  ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2004 ACS on STN
AN  2002:716967  CAPLUS
DN  137:231381
TI  Immunoglobulin control region
IN  Mundt, Cornelia Anna; Brueggemann, Marianne
PA  UK
SO  U.S. Pat. Appl. Publ., 21 pp.
    CODEN: USXXCO
DT  Patent
LA  English
FAN.CNT 1
    PATENT NO.      KIND  DATE         APPLICATION NO.  DATE
    --------------- ----  -------      ---------------  --------
PI  US 2002132373   A1    20020919     US 2002-81599    20020220
PRAI GB 2001-4299   A     20010221
AB  The region between  ***C*** . ***delta*** . and  ***C*** .
    ***gamma*** . ***3*** of ***IgH*** is unstable and may be a
    recombination hot spot. It has now been shown that 21 kb of the unstable
    region in the human ***IgH*** locus between ***C*** . ***delta***
    . and ***C*** . ***gamma*** . ***3*** contains a highly clustered
    array of a large no. of transcription factor-binding motifs interspersed
    with repeat sequences. Transfection assays revealed transcription
    enhancement and silencing activity at the pre B-cell stage and in
    transgenic mice strong enhancer function was identified in the bone
    marrow, the primary site of B-cell differentiation. Flow cytometry anal.
    of early B-cell populations showed that this enhancer is already active at
    the pro/pre B-cell stage where DNA rearrangement is initiated. The region
    accommodating E.delta.-.gamma.3 may exert locus control function at an
    early developmental stage, which may be crit. in normal and aberrant
    B-cell development.

L9  ANSWER 2 OF 2  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS
INC. on STN
AN  2001:300825  BIOSIS
DN  PREV200100300825
TI  t(5;14)(p15.3;q32) and t(14;19)(q32;p13.3): Novel, cryptic, recurring
    translocations involving the immunoglobulin heavy chain gene ( ***IGH***
    ) in B-cell non Hodgkin's lymphomas (NHL).
AU  Gozzetti, A. [Reprint author]; Davis, E. M. [Reprint author]; Espinosa,
    R., III [Reprint author]; Fernald, A. A. [Reprint author]; Anastasi, J.
    [Reprint author]; Le Beau, M. M. [Reprint author]
CS  Section of Hematology/Oncology, University of Chicago, Chicago, IL, USA
SO  Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 85a. print.
    Meeting Info.: 42nd Annual Meeting of the American Society of Hematology.
    San Francisco, California, USA. December 01-05, 2000. American Society of
    Hematology.
    CODEN: BLOOAW. ISSN: 0006-4971.
DT  Conference; (Meeting)
    Conference; Abstract; (Meeting Abstract)
LA  English
ED  Entered STN: 27 Jun 2001
    Last Updated on STN: 19 Feb 2002
AB  Chromosomal rearrangements involving the 5' ***IGH*** gene at 14q32 are
    observed in appnx75% of patients (pts) with B-cell NHL. This frequency
    may be underestimated, because the location of the 5' ***IGH*** locus
    within 8 kb of the telomeric repeats of 14q complicates the detection of
    translocations involving the telomeric band of partner chromosomes, since
    most terminal bands are G-negative with conventional staining techniques.
    To determine whether cryptic ***IGH*** translocations occur in NHL, we
    used dual color fluorescence in situ hybridization (FISH) of 5' and 3'
    ***IGH*** ***BAC*** clones: 158A2 (kindly provided by H.
    Avet-Loiseau, University Hospital, Nantes France) containing the JH and
    the 5' constant regions (Cpsiepsilon, Cgamma1, ***Cgamma3*** ,
    ***Cdelta*** , Cmu), and B200D12, a sub-telomeric probe 100-300 Kb from
    the telomeric repeats of 14q containing ***IGH*** variable sequences
    (provided by D.H. Ledbetter, Univ. of Chicago). We examined 51 pts with
```

B-cell NHL referred to our cytogenetics laboratory between 1989-1999, who had a normal karyotype (3 pts), clonal abnormalities not involving 14q32 (35 pts), or alterations of 14q32 other than recurring translocations, i.e., add(14)(q32) (13 pts). FISH detected 19 ***IGH*** translocations in 18/51 (35%) pts. Of the 13 pts with add(14)(q32), FISH identified the partner chromosome in 10 (77%) (3q27, 6 pts; 2p23, 19p13.3, 3p, 18q21, 1 pt each). 9/38 (24%) patients without visible alterations of 14q32 had masked (3 pts, 3q27) or cryptic ***IGH*** translocation (6 pts involving 5p15.3 (2 pts), 19p13.3 (2 pts), 3q27 (1 pt) or 3p (1 pt)). We identified two novel, recurring, cryptic translocations: t(5;14)(p15.3;q32) (2 pts) and t(14;19)(q32;p13.3) (3 pts). The 2 pts with a t(14;19) had small lymphocytic lymphocytic lymphoma/chronic lymphocytic leukemia (SLL/CLL) (CD5+), or diffuse large B cell lymphoma (DLBCL). The pt with a t(5;14) had a de novo CD5+ DLBCL. A pt with both t(14;19) and t(5;14) had DLBCL associated with CLL (CD5+). Thus, FISH results in the detection of cryptic or masked ***IGH*** rearrangements in apprx6% of all NHL cases, and apprx25% of those without visible rearrangements of 14q32.

=> s l7 and l3
L10 10 L7 AND L3

=> s l7 and l4
L11 2 L7 AND L4

=> s l10 or l11
L12 10 L10 OR L11

=> dup rem l12
PROCESSING COMPLETED FOR L12
L13 6 DUP REM L12 (4 DUPLICATES REMOVED)

=> d bib abs 1-
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L13 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:716967 CAPLUS
DN 137:231381
TI Immunoglobulin control region
IN Mundt, Cornelia Anna; Brueggemann, Marianne
PA UK
SO U.S. Pat. Appl. Publ., 21 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2002132373	A1	20020919	US 2002-81599	20020220
PRAI GB 2001-4299	A	20010221		

AB The region between ***C*** . ***delta*** . and ***C*** . ***gamma*** . ***3*** of ***IgH*** is unstable and may be a recombination hot spot. It has now been shown that 21 kb of the unstable region in the human ***IgH*** locus between ***C*** . ***delta*** . and ***C*** . ***gamma*** . ***3*** contains a highly clustered array of a large no. of transcription factor-binding motifs interspersed with repeat sequences. Transfection assays revealed transcription enhancement and silencing activity at the pre B-cell stage and in transgenic mice strong enhancer function was identified in the bone marrow, the primary site of B-cell differentiation. Flow cytometry anal. of early B-cell populations showed that this enhancer is already active at the pro/pre B-cell stage where DNA rearrangement is initiated. The region accommodating E.delta.-.gamma.3 may exert locus control function at an early developmental stage, which may be crit. in normal and aberrant B-cell development.

L13 ANSWER 2 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:300825 BIOSIS
DN PREV200100300825
TI t(5;14)(p15.3;q32) and t(14;19)(q32;p13.3): Novel, cryptic, recurring translocations involving the immunoglobulin heavy chain gene (***IGH***) in B-cell non Hodgkin's lymphomas (NHL).
AU Gozzetti, A. [Reprint author]; Davis, E. M. [Reprint author]; Espinosa, R., III [Reprint author]; Fernald, A. A. [Reprint author]; Anastasi, J. [Reprint author]; Le Beau, M. M. [Reprint author]
CS Section of Hematology/Oncology, University of Chicago, Chicago, IL, USA
SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 85a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 27 Jun 2001
Last Updated on STN: 19 Feb 2002
AB Chromosomal rearrangements involving the 5' ***IGH*** gene at 14q32 are observed in apprx75% of patients (pts) with B-cell NHL. This frequency may be underestimated, because the location of the 5' ***IGH*** locus within 8 kb of the telomeric repeats of 14q complicates the detection of translocations involving the telomeric band of partner chromosomes, since most terminal bands are G-negative with conventional staining techniques. To determine whether cryptic ***IGH*** translocations occur in NHL, we used dual color fluorescence in situ hybridization (FISH) of 5' and 3' ***IGH*** ***BAC*** clones: 158A2 (kindly provided by H. Avet-Loiseau, University Hospital, Nantes France) containing the JH and the 5' constant regions (Cpsiepsilon, Cgamma1, ***Cgamma3*** , ***Cdelta*** , Cmu), and B200D12, a sub-telomeric probe 100-300 Kb from the telomeric repeats of 14q containing ***IGH*** variable sequences (provided by D.H. Ledbetter, Univ. of Chicago). We examined 51 pts with B-cell NHL referred to our cytogenetics laboratory between 1989-1999, who had a normal karyotype (3 pts), clonal abnormalities not involving 14q32 (35 pts), or alterations of 14q32 other than recurring translocations, i.e., add(14)(q32) (13 pts). FISH detected 19 ***IGH*** translocations in 18/51 (35%) pts. Of the 13 pts with add(14)(q32), FISH identified the partner chromosome in 10 (77%) (3q27, 6 pts; 2p23, 19p13.3, 3p, 18q21, 1 pt each). 9/38 (24%) patients without visible alterations of 14q32 had masked (3 pts, 3q27) or cryptic ***IGH*** translocation (6 pts involving 5p15.3 (2 pts), 19p13.3 (2 pts), 3q27 (1 pt) or 3p (1 pt)). We identified two novel, recurring, cryptic translocations: t(5;14)(p15.3;q32) (2 pts) and t(14;19)(q32;p13.3) (3 pts). The 2 pts with a t(14;19) had small lymphocytic lymphocytic lymphoma/chronic lymphocytic leukemia (SLL/CLL) (CD5+), or diffuse large B cell lymphoma (DLBCL). The pt with a t(5;14) had a de novo CD5+ DLBCL. A pt with both t(14;19) and t(5;14) had DLBCL associated with CLL (CD5+). Thus, FISH results in the detection of cryptic or masked ***IGH*** rearrangements in apprx6% of all NHL cases, and apprx25% of those without visible rearrangements of 14q32.

L13 ANSWER 3 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 1
AN 2000:25549 BIOSIS
DN PREV200000025549
TI B-cell tumorigenesis in mice carrying a yeast ***artificial*** ***chromosome*** -based immunoglobulin heavy/c-myc translocus is independent of the heavy chain intron enhancer (Emu).
AU Palomo, Concepcion; Zou, Xiangang; Nicholson, Ian C.; Butzler, Christoph; Bruggemann, Marianne [Reprint author]
CS Laboratory of Developmental Immunology, Department of Development and Genetics, Babraham Institute, Babraham, Cambridge, CB2 4AT, UK
SO Cancer Research, (Nov. 1, 1999) Vol. 59, No. 21, pp. 5625-5628. print.
CODEN: CNREA8. ISSN: 0008-5472.
DT Article
LA English
ED Entered STN: 29 Dec 1999
Last Updated on STN: 31 Dec 2001
AB We have used ***YAC*** (yeast ***artificial*** ***chromosome***) technology to create large translocation regions where the c-myc proto-oncogene is coupled to the core region of the human immunoglobulin heavy chain (***IgH***) locus (from VH2-5 through to ***Cdelta***). Chimeric mice were obtained from embryonic stem cells carrying a single copy of the 240-kb ***IgH*** /c-myc translocation region. B-cell tumorigenesis occurs in the translocus mice, even when the entire Emu intron enhancer region between the joining segments and switch mu is deleted. This demonstrates that as yet unidentified regulatory elements in the ***IgH*** locus, independent from the known enhancers, are sufficient to cause B-cell specific activation of c-myc after translocation. The phenotype of tumors from ***IgH*** /c-myc ***YAC*** transgenic mice with or without Emu (B220+, IgM+/IgD+) is reminiscent of Burkitt's lymphoma. A rapidly expanding abnormal B-cell population is present at birth and accumulates in bone marrow, periphery, and spleen, well before discrete tu mor establishment. Molecular analysis identified a clonal origin, with rearrangement of one mouse heavy chain allele retained in tumor cells from different sites, whereas subsequent rearrangements of heavy or light chain loci can be diverse. These mice routinely develop mature B-cell tumors early in life and may provide an invaluable resource of a B-cell lymphoma model.

L13 ANSWER 4 OF 6 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 2000213160 EMBASE
TI Antibody engineering via genetic engineering of the mouse: XenoMouse strains are a vehicle for the facile generation of therapeutic human monoclonal antibodies.
AU Green L.L.
CS L.L. Green, Abgenix, 7601 Dumbarton Circle, Fremont, CA 94555, United States
SO Journal of Immunological Methods, (1999) 231/1-2 (11-23).
Refs: 50
ISSN: 0022-1759 CODEN: JIMMBG
PUI S 0022-1759(99)00137-4
CY Netherlands
DT Journal; Article
FS 026 Immunology, Serology and Transplantation
037 Drug Literature Index
LA English
SL English
AB The major impediment to the development of murine monoclonal antibodies (mAbs) for therapy in humans has been the difficulty in reducing their potential immunogenicity. XenoMouse(TM) mice obviate this problem while retaining the relative ease of generating mAbs from a mouse. XenoMouse strains include germline-configured, megabase-sized YACs carrying portions

of the human ***IgH*** and Ig.kappa. loci, including the majority of the variable region repertoire, the genes for C.mu., ***C*** . ***delta*** . and either C.gamma.1, C.gamma.2, or C.gamma.4, as well as the cis elements required for their function. The ***IgH*** and Ig.kappa. transgenes were bred onto a genetic background deficient in production of murine immunoglobulin. The large and complex human variable region repertoire encoded on the Ig transgenes in XenoMouse strains support the development of large peripheral B cell compartments and the generation of a diverse primary immune repertoire similar to that from adult humans. Immunization of XenoMouse mice with human antigens routinely results in a robust secondary immune response, which can ultimately be captured as a large panel of antigen-specific fully human IgG.kappa. mAbs of sub-nanomolar affinities. Monoclonal antibodies from XenoMouse animals have been shown to have therapeutic potential both in vitro and in vivo, and appear to have the pharmacokinetics of normal human antibodies based on human clinical trials. The utility of XenoMouse strains for the generation of large panels of high- affinity, fully human mAbs can be made available to researchers in the academic and private sectors, and should accelerate the development and application of mAbs as therapeutics for human disease. (C) 1999 Elsevier Science B.V.

L13 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:568935 CAPLUS
DN 129:187955
TI Yeast ***artificial*** ***chromosomes*** for studying aberrant proto-oncogene expression after translocation to complex loci, especially immunoglobulin loci
IN Bruggemann, Marianne
PA The Babraham Institute, UK
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9836082	A1	19980820	WO 1998-GB433	19980212
W: AU, GB, JP, US				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9860011	A1	19980908	AU 1998-60011	19980212
PRAI GB 1997-2802		19970212		
WO 1998-GB433		19980212		

AB A method of studying the effects of cis-regulatory elements on the expression of translocated protooncogenes using yeast ***artificial*** ***chromosomes*** is described. The method uses yeast ***artificial*** ***chromosomes*** (YACs) to integrate large, complex, genes and the protooncogene into a suitable host. The method is particularly intended to study regulatory elements of Ig loci, esp. the ***IgH*** gene, on the expression of protooncogenes, esp. the c-myc gene translocated in Burkitt's lymphoma. A ***YAC*** contg. 220 kb of the ***IgH*** locus including a large segment of the V-D-J region, the E.mu., C.mu. and ***C*** . ***delta*** . regions with a c-myc gene downstream of the ***C*** . ***delta*** . region was constructed and introduced in embryonic stem cells that were used to construct transgenic mice. The mice developed soft tumors in many tissues leading to visible signs of illness 7-8 wk after birth. Cells from these tumors resembled common lymphoid cell lines and were shown to be monoclonal B cell-derived lines.
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 6 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 2
AN 1995:252599 BIOSIS
DN PREV199598266899
TI Analysis of the Structural Integrity of YACs Comprising Human Immunoglobulin Genes in Yeast and in Embryonic Stem Cells.
AU Mendez, Michael J.; Abderrahim, Hadi; Noguchi, Masato; David, Nathaniel E.; Hardy, Margaret C.; Green, Larry L.; Tsuda, Hirohisa; Yoast, Sienna; Maynard-Currie, Catherine E.; Garza, Dan; Gemmill, Robert; Jakobovits, Aya; Klapholz, Sue [Reprint author]
CS Cell Genesys Inc., 322 Lakeside Drive, Foster City, CA 94404, USA
SO Genomics, (1995) Vol. 26, No. 2, pp. 294-307.
CODEN: GNMCEP. ISSN: 0888-7543.
DT Article
LA English
ED Entered STN: 13 Jun 1995
Last Updated on STN: 13 Jun 1995
AB With the goal of creating a strain of mice capable of producing human antibodies, we are cloning and reconstructing the human immunoglobulin germline repertoire in yeast ***artificial*** ***chromosomes*** (YACs). We describe the identification of YACs containing variable and constant region sequences from the human heavy chain (***IgH***) and kappa light chain (IgK) loci and the characterization of their integrity in yeast and in mouse embryonic stem (ES) cells. The ***IgH*** locus-derived ***YAC*** contains five variable (V-H) genes, the major diversity (D) gene cluster, the joining (J-H) genes, the intronic enhancer (E-H), and the constant region genes, mu (C-mu) and delta (***C*** - ***delta***). Two IgK locus-derived YACs each contain three variable (V-kappa) genes, the joining (J-kappa) region, the intronic enhancer (E-kappa), the constant gene (C-kappa), and the kappa deleting element (kde). The ***IgH*** ***YAC*** was unstable in yeast, generating a variety of deletion derivatives, whereas both IgK YACs were stable. YACs encoding heavy chain and kappa light chain, retrofitted with the mammalian selectable marker, hypoxanthine phosphoribosyltransferase (HPRT), were each introduced into HPRT-deficient mouse ES cells. Analysis of ***YAC*** integrity in ES cell lines revealed that the majority of DNA inserts were integrated in substantially intact form.

=> d his

(FILE 'HOME' ENTERED AT 18:00:42 ON 10 MAR 2004)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 18:01:04 ON 10 MAR 2004
L1 11819 S ARTIFICIAL CHROMOSOM?
L2 7110 S IGH
L3 6449 S C DELTA OR CDELTA
L4 424 S CGAMMA3 OR C GAMMA 3
L5 67 S L1 AND L2
L6 25475 S L1 OR BAC OR YAC
L7 116 S L6 AND L2
L8 2 S L7 AND L3 AND L4
L9 2 DUP REM L8 (0 DUPLICATES REMOVED)
L10 10 S L7 AND L3
L11 2 S L7 AND L4
L12 10 S L10 OR L11
L13 6 DUP REM L12 (4 DUPLICATES REMOVED)

=> FIL STNGUIDE

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=> FIL BIOSIS EMBASE CAPLUS

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FULL ESTIMATED COST		0.06	54.52

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	ENTRY	SINCE FILE SESSION	TOTAL
CA SUBSCRIBER PRICE		0.00	-2.08

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FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 18:01:04 ON 10 MAR 2004
L1 11819 S ARTIFICIAL CHROMOSOM?
L2 7110 S IGH
L3 6449 S C DELTA OR CDELTA
L4 424 S CGAMMA3 OR C GAMMA 3
L5 67 S L1 AND L2
L6 25475 S L1 OR BAC OR YAC
L7 116 S L6 AND L2
L8 2 S L7 AND L3 AND L4
L9 2 DUP REM L8 (0 DUPLICATES REMOVED)
L10 10 S L7 AND L3
L11 2 S L7 AND L4
L12 10 S L10 OR L11
L13 6 DUP REM L12 (4 DUPLICATES REMOVED)

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FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 18:12:01 ON 10 MAR 2004

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ENTRY SESSION
CA SUBSCRIBER PRICE 0.00 -2.08

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TERMINAL (ENTER 1, 2, 3, OR ?):2

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NEWS 6 OCT 10 PCTFULL: Two new display fields added
NEWS 7 OCT 21 BIOSIS file reloaded and enhanced
NEWS 8 OCT 28 BIOSIS file segment of TOXCENTER reloaded and enhanced
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NEWS 14 DEC 17 DGENE: Two new display fields added
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NEWS 16 DEC 19 CROPU no longer updated; subscriber discount no longer available
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NEWS 18 DEC 22 IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields
NEWS 19 DEC 22 ABI-INFORM now available on STN
NEWS 20 JAN 27 Source of Registration (SR) information in REGISTRY updated and searchable
NEWS 21 JAN 27 A new search aid, the Company Name Thesaurus, available in CA/CAplus
NEWS 22 FEB 05 German (DE) application and patent publication number format changes
NEWS 23 MAR 03 MEDLINE and LMEDLINE reloaded
NEWS 24 MAR 03 MEDLINE file segment of TOXCENTER reloaded
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L1 25488 ARTIFICIAL CHROMOSOME? OR YAC OR BAC

=> s l1 and alu repeats
L2 17 L1 AND ALU REPEATS

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L3 6 DUP REM L2 (11 DUPLICATES REMOVED)

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L3 ANSWER 1 OF 6 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 1
AN 2004079206 EMBASE
TI Closing the gaps on human chromosome 19 revealed genes with a high density of repetitive tandemly arrayed elements.
AU Leem S.-H.; Kouprina N.; Grimwood J.; Kim J.-H.; Mullokandov M.; Yoon Y.-H.; Chae J.-Y.; Morgan J.; Lucas S.; Richardson P.; Detter C.; Glavina T.; Rubin E.; Barrett J.C.; Larionov V.
CS V. Larionov, Laboratory of Biosystem and Cancer, Center for Cancer Research, National Cancer Institute NCI NIH, Bethesda, MD 20892, United States. larionov@mail.nih.gov
SO Genome Research, (2004) 14/2 (239-246).
Refs: 27
ISSN: 1088-9051 CODEN: GEREFS
CY United States
DT Journal; Article
FS 022 Human Genetics
LA English
SL English
AB The reported human genome sequence includes about 400 gaps of unknown sequence that were not found in the bacterial ***artificial*** ***chromosome*** (***BAC***) and cosmid libraries used for sequencing of the genome. These missing sequences correspond to .apprx.1% of euchromatic regions of the human genome. Gap filling is a laborious process because it relies on analysis of random clones of numerous genomic ***BAC*** or cosmid libraries. In this work we demonstrate that closing the gaps can be accelerated by a selective recombinational capture of missing chromosomal segments in yeast. The use of both methodologies allowed us to close the four remaining gaps on the human chromosome 19. Analysis of the gap sequences revealed that they contain several abnormalities that could result in instability of the sequences in microbe hosts, including large blocks of micro- and minisatellites and a high density of ***Alu*** ***repeats*** . Sequencing of the gap regions, in both ***BAC*** and ***YAC*** forms, allowed us to generate a complete sequence of four genes, including the neuronal cell signaling gene SCK1/SLI. The SCK1/SLI gene contains a record number of minisatellites, most of which are polymorphic and transmitted through meiosis following a Mendelian inheritance. In conclusion, the use of the alternative recombinational cloning system in yeast may greatly accelerate work on closing the remaining gaps in the human genome (as well as in other complex genomes) to achieve the goal of annotation of all human genes.

L3 ANSWER 2 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 2
AN 1999:236611 BIOSIS
DN PREV199900236611
TI Organization of the gene for gelatin-binding protein (GBP28).
AU Saito, Kiyomi; Tobe, Takashi [Reprint author]; Minoshima, Shinsei; Asakawa, Shuichi; Sumiya, Junichi; Yoda, Madoka; Nakano, Yasuko; Shimizu, Nobuyoshi; Tomita, Motowo
CS Department of Medicinal Information, School of Pharmaceutical Sciences, Showa University, 1-5-8, Hatanodai, Shinagawa-ku, Tokyo, Japan
SO Gene (Amsterdam), (March 18, 1999) Vol. 229, No. 1-2, pp. 67-73. print.
CODEN: GENED6. ISSN: 0378-1119.
DT Article
LA English
OS Genbank-AB012163; EMBL-AB012163; DDBJ-AB012163; Genbank-AB012164;
EMBL-AB012164; DDBJ-AB012164; Genbank-AB012165; EMBL-AB012165; DDBJ-AB012165

ED Entered STN: 17 Jun 1999
Last Updated on STN: 17 Jun 1999
AB GBP28 is a novel human plasma gelatin-binding protein that is encoded by apM1 mRNA, expressed specifically in adipose tissue. Three overlapping clones (two lambda clones and one ***BAC*** clone) containing the human plasma gelatin-binding protein (GBP28) gene were isolated and characterized. The GBP28 gene spans 16 kb and is composed of three exons from 18 bp to 4277 bp in size with consensus splice sites. The sizes of the two introns were 0.8 and 12 kb, respectively. The gene's regulatory sequences contain putative promoter elements, but no typical TATA box. The third exon of this gene contains a long 3'-untranslated sequence containing three ***Alu*** ***repeats*** . The exon-intron organization of this gene was very similar to that of obese gene, encoding leptin. We also report the chromosome mapping of this gene by fluorescence in situ hybridization (FISH) using a genomic DNA fragment as a probe. The GBP28 gene was located on human chromosome 3q27. The nucleotide sequence data reported in this paper will appear in the DDBJ/EMBL/GenBank nucleotide sequence databases with the accession numbers
ABO12163, ABO12164 or ABO12165.

L3 ANSWER 3 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 3
AN 1997:344969 BIOSIS
DN PREV199799644172
TI Physical mapping 220 kb centromeric of the human MHC and DNA sequence analysis of the 43-kb segment including the RING1, HKE6, and HKE4 genes.
AU Kikuti, Yara Yukie; Tamiya, Gen; Ando, Asako; Chen, Lei; Kimura, Minoru; Ferreira, Euripides; Tsuji, Kimiyoshi; Trowsdale, John; Inoko, Hidetoshi [Reprint author]
CS Dep. Mol. Life Sci., Tokai Univ. Sch. Med., Bohseidai, Isehara, Kanagawa 259-11, Japan
SO Genomics, (1997) Vol. 42, No. 3, pp. 422-435.
CODEN: GNMCEP. ISSN: 0888-7543.
DT Article
LA English
ED Entered STN: 11 Aug 1997
Last Updated on STN: 11 Aug 1997
AB A cosmid contig was constructed from a ***YAC*** clone with a 220-kb insert that spans the centromeric side of the human MHC class II region, corresponding to the mouse t complex. The gene order was identified to be HSET-HKE1.5-HKE2-HKE3-RING1-HKE6-HKE4 (RING5). The genomic sequence of a
42,801-bp long region encoded by one cosmid clone in the RING1, HKE6, and HKE4 subregions was determined by the shotgun method. The exon-intron organization of these three genes, RING1 (Ring finger protein), HKE6 (steroid dehydrogenase-like protein), and HKE4 (transmembrane protein with histidine-rich charge clusters), was determined. The previously reported RING2 gene was revealed to be identical to HKE6. Transcripts from HKE4 were detected in the placenta, lung, kidney, and pancreas. Those of HKE6 were found in the liver and pancreas. The 25-kb region proximal to the RING1 gene includes an extensive dense cluster of ***Alu*** ***repeats*** (about 1.2 Alu per kb), and no gene has been identified in this so far. The region is equivalent to part of the mouse t complex and could be of relevance to human development.

L3 ANSWER 4 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 4
AN 1997:19866 BIOSIS
DN PREV199799319069
TI Highly selective isolation of human DNAs from rodent-human hybrid cells as circular yeast ***artificial*** ***chromosomes*** by transformation-associated recombination cloning.
AU Larionov, Vladimir [Reprint author]; Kouprina, Natalya; Graves, Joan; Resnick, Michael A.
CS Inst. Cytol., Russian Academy Sci., St. Petersburg 194064, Russia
SO Proceedings of the National Academy of Sciences of the United States of America, (1996) Vol. 93, No. 24, pp. 13925-13930.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 15 Jan 1997
Last Updated on STN: 15 Jan 1997
AB Transformation-associated recombination (TAR) can be exploited in yeast to clone human DNAs. TAR cloning was previously accomplished using one or two telomere-containing vectors with a common human repeat(s) that could recombine with human DNA during transformation to generate yeast ***artificial*** ***chromosomes*** (YACs). On basis of the proposal that broken DNA ends are more recombinogenic than internal sequences, we have investigated if TAR cloning could be applied to the generation of circular YACs by using a single centromere vector containing various human repeats at opposite ends. Transformation with these vectors along with human DNA led to the efficient isolation of circular YACs with a mean size of apprxeq 150 kb. The circular YACs are stable and they can be easily separated from yeast chromosomes or moved into bacterial cells if the TAR vector contains an Escherichia coli F-factor cassette. More importantly, circular TAR cloning enabled the selective isolation of human DNAs from monochromosomal human-rodent hybrid cell lines. Although lt 2% of the DNA in the hybrid cells was human, as much as 80% of transformants had human DNA YACs when a TAR cloning vector contained ***Alu*** ***repeats*** . The level of enrichment of human DNA was nearly 3000-fold. A comparable level of enrichment was demonstrated with DNA isolated from a radiation hybrid cell line containing only 5 Mb of human DNA. A high selectivity of human DNA cloning was also observed for linear TAR cloning with two telomere vectors. No human-rodent chimeras were detected among YACs generated by TAR cloning. The results with a circular TAR cloning vector or two vectors differed from results with a single-telomere vector in that the latter often resulted in a series of terminal deletions in linear YACs. This could provide a means for physical mapping of cloned material.

L3 ANSWER 5 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 5
AN 1995:18064 BIOSIS
DN PREV199598032364
TI Recombination during transformation as a source of chimeric mammalian ***artificial*** ***chromosomes*** in yeast (YACs).
AU Larionov, V. [Reprint author]; Kouprina, N.; Nikolaishvili, N.; Resnick, M. A.
CS Inst. Cytol., Russian Acad. Sci., St. Petersburg, Russia
SO Nucleic Acids Research, (1994) Vol. 22, No. 20, pp. 4154-4162.
CODEN: NARHAD. ISSN: 0305-1048.
DT Article
LA English
ED Entered STN: 11 Jan 1995
Last Updated on STN: 11 Jan 1995
AB Mammalian DNAs cloned as ***artificial*** ***chromosomes*** in yeast (YACs) frequently are chimeras formed between noncontiguous DNAs. Using pairs of human and mouse YACs we examined the contribution of recombination during transformation or subsequent mitotic growth to chimeric ***YAC*** formation. The DNA from pairs of yeast strains containing homologous or heterologous YACs was transformed into a third strain under conditions typical for the development of ***YAC*** libraries. One ***YAC*** was selected and the presence of the second was then determined. Co-penetration of large molecules, as deduced from co-transformation of markers identifying the different YACs, was gt 50%. In approximately half the cells receiving two homologous YACs, the YACs had undergone recombination. Co-transformation depends on recombination since it was reduced nearly 10-fold when the YACs were heterologous. While mitotic recombination between homologous YACs is nearly 100-fold higher than for yeast chromosomes, the level is still much lower than observed during transformation. To investigate the role of commonly occurring ***Alu*** ***repeats*** in chimera formation, spheroplasts were transformed with various human YACs and an unselected DNA fragment containing an Alu at one end and a telomere at the other. When unbroken YACs were used, between 1 and 6% of the selected YACs could
incorporate the fragment as compared to 49% when the YACs were broken. We
propose that Alu's or other commonly occurring repeats could be an important source of chimeric YACs. Since the frequency of chimeras formed between YACs or a ***YAC*** and an Alu-containing fragment was reduced when a rad52 mutant was the recipient and since intraYAC deletions are reduced, rad52 and possibly other recombination-deficient mutants are expected to be useful for ***YAC*** library development.

L3 ANSWER 6 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 6
AN 1989:472179 BIOSIS
DN PREV198988107939; BA88:107939
TI ALU POLYMERASE CHAIN REACTION A METHOD FOR RAPID ISOLATION OF
HUMAN-SPECIFIC SEQUENCES FROM COMPLEX DNA SOURCES.
AU NELSON D L [Reprint author]; LEDBETTER S A; CORBO L; VICTORIA M F; RAMIREZ-SOLIS R; WEBSTER T D; LEDBETTER D H; CASKEY C T
CS HOWARD HUGHES MED INST, BAYLOR COLL MED, ONE BAYLOR PLAZA, TEXAS 77030,
USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1989) Vol. 86, No. 17, pp. 6686-6690.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 17 Oct 1989
Last Updated on STN: 17 Oct 1989
AB Current efforts to map the human genome are focused on individual chromosomes or smaller regions and frequently rely on the use of somatic cell hybrids. We report the application of the polymerase chain reaction to direct amplification of human DNA from hybrid cells containing regions of the human genome in rodent cell backgrounds using primers directed to the human Alu repeat element . We demonstrate Alu-directed amplification of a fragment of the human HPRT gene from both hybrid cell and cloned DNA and identify through sequence analysis the ***Alu*** ***repeats*** involved in this amplification. We also demonstrate the application of this technique to identify the chromosomal locations of large fragments of the human X chromosome cloned in a yeast ***artificial*** ***chromosome*** and the general applicability of the method to the preparation of DNA probes from cloned human sequences. The technique allows rapid gene mapping and provides a simple method for the isolation and analysis of specific chromosomal regions.

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'E47' NOT FOUND
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L4 170 L1 AND (IKAROS OR OCT-1 OR USF OR MYC OR CEBPBETA)

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L6 ANSWER 1 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:223833 BIOSIS
DN PREV200200223833
TI Molecular cloning and chromosomal localization of the murine H-K-ATPase alpha2 subunit gene.
AU Kone, Bruce C. [Reprint author]; Kuncewicz, Teresa [Reprint author]
CS Division of Renal Diseases and Hypertension, University of Texas Medical School-Houston, Houston, TX, USA
SO Journal of the American Society of Nephrology, (September, 2000) Vol. 11, No. Program and Abstract Issue, pp. 31A. print.
Meeting Info.: 33rd Annual Meeting of the American Society of Nephrology and the 2000 Renal Week. Toronto, Ontario, Canada. October 10-16, 2000. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 3 Apr 2002
Last Updated on STN: 3 Apr 2002
AB The H-K-ATPase alpha2 (HKalpha2) subunit plays important roles in sodium, potassium and acid-base homeostasis. Mice genetically deficient in this gene exhibit fecal potassium wasting and profound hypokalemia during dietary potassium deprivation. The HKalpha2 gene is principally expressed in kidney and colon and is differentially regulated by aldosterone and potassium restriction in the two organs. Despite this evidence for complex regulatory controls, nothing is known of the genomic structure, organization or chromosomal localization of the HKalpha2 gene. To gain insights into structure-function aspects of the gene, we used an HKalpha2-specific cDNA probe to screen an 129/SvJ mouse genomic library by filter hybridization. A clone corresponding to 15 kB of the 5 prime-flanking sequence extending into intron 8 of the HKalpha2 gene was isolated. 2.6 kB of the proximal promoter region was sequenced and revealed multiple potential cis-elements, including a GRE and sites for CREB/ATF, AP-1, Sp1, ***Oct*** - ***1*** , NF-kappaB, C/EBP, and gut-enriched Kruppel-like factor. Fluorescence in situ hybridization analysis of normal metaphase chromosomes showed cohybridization of the HKalpha2 genomic probe with a probe specific for the centromeric region of chromosome 14, mapping the HKalpha2 gene to mouse chromosome band 14C3.
The entire HKalpha2 gene was then cloned from a murine ***BAC*** library using PCR generated HKalpha2-specific cDNA probes and filter hybridization. We have thus far sequenced the first 8 exons and mapped the intron/exon boundaries of this region. Since gene control elements may be positioned throughout the gene, this clone will allow detailed analysis of HKalpha2 gene transcription in vitro and in vivo. Moreover it will allow direct structural comparison to the putative HKalpha2 ortholog, human ATPIAL1.

L6 ANSWER 2 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:300829 BIOSIS
DN PREV200100300829
TI The t(6;14)(p21;q32) translocation causes dysregulation of cyclin D3 in multiple myeloma.
AU Qi, Ying [Reprint author]; Gabrea, Ana [Reprint author]; Sawyer, Jeffrey; Shaughnessy, John D., Jr.; Barlogie, Bart; Bergsagel, P. Leif; Kuehl, W. Michael [Reprint author]
CS Genetics Department, Medicine Branch, National Cancer Institute, Bethesda, MD, USA
SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 86a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 27 Jun 2001
Last Updated on STN: 19 Feb 2002
AB Reciprocal translocations involving the IgH locus (14q32) appear to provide an early transformation event in most multiple myeloma (MM) tumors. These primary translocations are targeted to V/D/J sequences due to errors in VDJ recombination or somatic hypermutation, or to IgH switch regions due to errors in switch recombination. The translocations include a promiscuous array of at least 20 nonrandom chromosomal partners, each including an oncogene that is dysregulated by juxtaposition to potent IgH enhancers. The most frequent partners include: 11q13(cyclin D1), 4p16.3 (FGFR3 and MM.SET), and 16q23 (c-maf), which together are present in approximately 50% of MM tumors. Cyclin D3 is expressed at similar levels in 25 MM cell lines, except for one cell line that expresses nearly ten times as much cyclin D3 mRNA and protein as any other cell line. In this MM cell line, FISH analyses confirmed the presence of a t(6;14)(p21;q32) translocation, with close juxtaposition of cyclin D3 and IgH Ealpha enhancer sequences. The cloned translocation breakpoint junction occurs within a gamma4 IgH switch region and includes 6p21 sequences that co-localize with cyclin D3 sequences on a ***BAC*** clone. In screening a panel of 30 MM tumors, we identified a single tumor with a t(6;22)(p21;q11) translocation, with cyclin D3 and Clambda enhancer sequences juxtaposed on der(6). As a result the IgH and Iglambda translocations bracket the cyclin D3 gene, similar to results obtained for other genes (c- ***myc*** , c-maf) dysregulated by Ig translocations. From conventional cytogenetic and/or SKY analyses of several hundred MM tumors, the t(6;14)(p21;q32) translocation is present in about 2-3% of tumors. Additional FISH analyses of metaphase chromosomes confirm that: 1) cyclin D3 and IgH Ealpha enhancer sequences are juxtaposed in 4 MM tumors with t(6;14)(p21;q32); and 2) in at least 3 of these tumors the ***BAC*** FISH probe described above identifies both der(14) and der(16), indicating that the breakpoints occur no more than 200 kb centromeric to cyclin D3. We hypothesize that primary Ig translocations which dysregulate either cyclin D1 or cyclin D3 promote the transformation of quiescent long-lived plasma cells into proliferating MGUS tumor cells.

L6 ANSWER 3 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:118346 BIOSIS
DN PREV200100118346
TI Attenuation of ototoxic effects of cisplatin on inner ear cells by HSV vector NT-3 transduction.
AU Chen, X. [Reprint author]; Frisina, R. D.; Bowers, W. J.; Frisina, D. R.; Federoff, H. J.
CS U. Rochester Med. Sch., Rochester, NY, USA
SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-826.7. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 7 Mar 2001
Last Updated on STN: 15 Feb 2002
AB Ototoxicity is a major dose-limiting side effect of cisplatin (DDP) treatment. It can cause destruction of cochlear cells and neurons. As Trkc expressing spiral ganglion neurons (SGN) may be protected from DDP by localized expression of NT-3, we developed a HSV amplicon vector to transduce a c- ***myc*** tagged version of human NT-3. In the HSVCMVnt-3myc vector the chimeric neurotrophin cDNA was placed under the transcriptional control of the CMV IE promoter, thus generating an active transcription unit. The resultant vector was packaged with the helper virus-free method utilizing ***BAC*** , resulting in titers exceeding 5x6E10 ip/ml. Vector stocks were tested in vitro for their capacity to direct expression of NT-3 mRNA and gene product. Transduction of primary SGNs at an MOI of 0.23-0.28 bfu/ml resulted in production of 3ng/ml NT-3 over 48 hours. To determine whether NT-3 overexpression would abrogate DDP toxicity, mouse cochlear explants in culture were transduced with HSVCMVnt-3myc or control HSVCMVmiap (expressing the reporter gene murine
intestinal alkaline phosphatase only) 48 hours prior to treatment with DDP (4ig/ml). After an additional 96 hours in organotypic culture, cochlear explants were analyzed for surviving SGNs and hair cells. In cultures transduced with HSVCMVnt-3myc, a significantly greater number of hair cells and SGNs survived than in controls. These data show that helper-free virus transduction with neurotrophic factor genes attenuates the ototoxic action of DDP. The utility of the approach for in vivo protection is under study.

L6 ANSWER 4 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2000:346542 BIOSIS
DN PREV200000346542
TI Isolation and characterization of the human UGT2B7 gene.
AU Carrier, Jean-Sebastien; Turgeon, David; Journault, Kim; Hum, Dean W.; Belanger, Alain [Reprint author]
CS Laboratory of Molecular Endocrinology, CHUL Research Center, 2705 Laurier Blvd., Quebec, G1V 4G2, Canada
SO Biochemical and Biophysical Research Communications, (June 7, 2000) Vol. 272, No. 2, pp. 616-621. print.
CODEN: BBRCA9. ISSN: 0006-291X.
DT Article
LA English
ED Entered STN: 16 Aug 2000
Last Updated on STN: 7 Jan 2002
AB Glucuronidation is a major pathway involved in the metabolism of drugs and numerous endogenous compounds, such as bile acids and steroid hormones. The enzymes responsible for this conjugation reaction are UDP-glucuronosyltransferases (UGT). Among the UGT2B subfamily, UGT2B7, a

UGT enzyme present in the liver and several steroid target tissues, is an important member since it conjugates a large variety of compounds including estrogens, androgens, morphine, AZT, and retinoic acid. Although this enzyme is well characterized, the gene encoding the UGT2B7 protein and its promoter region remain unknown. In this article, we report the genomic organization and the promoter region of the human UGT2B7 gene. To isolate this gene, a P-1 ***artificial*** ***chromosome*** (PAC) library was screened with a full length UGT2B7 probe and a clone of approximately 100 kb in length was isolated. In addition to the UGT2B7 gene, this PAC contains two other UGT2B genes previously characterized, namely UGT2B26P and UGT2B27P. The UGT2B7 gene
is composed of six exons spanning approximately 16 kb, with introns ranging from 0.7 to 4.2 kb. The 5'-flanking region of the human UGT2B7 gene contains several potential cis-acting elements such as ***Oct*** - ***1*** , Pbx-1, and C/EBP. Only one TATA-box at nucleotide - 106 was found within the first 500 nucleotides relative to the adenine base of the initiator ATG codon. Characterization of the UGT2B7 gene provides insight into the organization and regulation of this important metabolic gene.

L6 ANSWER 5 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2000:190939 BIOSIS
DN PREV200000190939
TI Microdissection and FISH investigations in acute myeloid leukemia: A step forward to full identification of complex karyotypic changes.
AU Falzetti, Daniela; Vermeesch, Joris R.; Matteucci, Caterina; Ciolli, Stefania; Martelli, Massimo F.; Marynen, Peter; Mecucci, Cristina [Reprint author]
CS Hematology and Bone Marrow Transplantation, Policlinico Monteluce, Via Brunamonti, 06100, Perugia, Italy
SO Cancer Genetics and Cytogenetics, (April 1, 2000) Vol. 118, No. 1, pp. 28-34. print.
CODEN: CGCYDF. ISSN: 0165-4608.
DT Article
LA English
ED Entered STN: 17 May 2000
Last Updated on STN: 4 Jan 2002
AB Complex chromosomal rearrangements in malignant hemopathies frequently remain unclarified because of paucity of material for further fluorescence in situ hybridization analyses and/or lack of suitable probes. Chromosome microdissection (MD) can be an adequate approach to elucidate chromosome aberrations unrecognizable by conventional karyotyping. We applied MD in two patients with acute myeloid leukemia (AML) and unidentified chromosome changes at karyotype. Microdissection of a ring chromosome in an AML-M5 case revealed 21q polysomy. In an AML-M4 case, MD of an add(15p) disclosed a t(8;15) with over-representation of both 8q22 and 8q24 bands. ***YAC*** probes were helpful in showing duplication of the ETO gene at 8q22, and amplification of C- ***MYC*** , at 8q24.

L6 ANSWER 6 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2000:113766 BIOSIS
DN PREV200000113766
TI Isolation and characterization of the human UGT2B15 gene, localized within a cluster of UGT2B genes and pseudogenes on chromosome 4.
AU Turgeon, David; Carrier, Jean-Sebastien; Levesque, Eric; Beatty, Barbara G.; Belanger, Alain [Reprint author]; Hum, Dean W. [Reprint author]
CS Laboratory of Molecular Endocrinology, Laval University, Laval, Quebec, G1V 4G2, Canada
SO Journal of Molecular Biology, (Jan. 21, 2000) Vol. 295, No. 3, pp. 489-504. print.
CODEN: JMOBAK. ISSN: 0022-2836.
DT Article
LA English
ED Entered STN: 29 Mar 2000
Last Updated on STN: 3 Jan 2002
AB Glucuronidation is a major pathway of androgen metabolism and is catalyzed by UDP-glucuronosyltransferase (UGT) enzymes. UGT2B15 and UGT2B17 are 95%
identical in primary structure, and are expressed in steroid target tissues where they conjugate C19 steroids. Despite the similarities, their regulation of expression are different; however, the promoter region and genomic structure of only the UGT2B17 gene have been characterizedX to date. To isolate the UGT2B15 gene and other novel steroid-conjugating UGT2B genes, eight P-1-derived ***artificial*** ***chromosomes*** (PAC) clones varying in length from 30 kb to 165 kb were isolated. The entire UGT2B15 gene was isolated and characterized from the PAC clone 21598 of 165 kb. The UGT2B15 and UGT2B17 genes are highly conserved, are
both composed of six exons spanning approximately 25 kb, have identical exon sizes and have identical exon-intron boundaries. The homology between the two genes extend into the 5'-flanking region, and contain several conserved putative cis-acting elements including Pbx-1, C/EBP, AP-1, ***Oct*** - ***1*** and NF/kappaB. However, transfection studies revealed differences in basal promoter activity between the two genes, which correspond to regions containing non-conserved potential elements. The high degree of homology in the 5'-flanking region between the two genes is lost upstream of -1662 in UGT2B15, and suggests a site of genetic recombination involved in duplication of UGT2B genes. Fluorescence in situ hybridization mapped the UGT2B15 gene to chromosome 4q13.3-21.1. The other PAC clones isolated contain exons from the UGT2B4, UGT2B11 and UGT2B17 genes. Five novel exons, which are highly homologous
to the exon 1 of known UGT2B genes, were also identified; however, these exons contain premature stop codons and represent the first recognized pseudogenes of the UGT2B family. The localization of highly homologous UGT2B genes and pseudogenes as a cluster on chromosome 4q13 reveals the complex nature of this gene locus, and other novel homologous UGT2B genes encoding steroid conjugating enzymes are likely to be found in this region of the genome.

L6 ANSWER 7 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2000:61885 BIOSIS
DN PREV200000061885
TI Human Aiolos, an ***Ikaros*** -related zinc finger DNA binding protein: cDNA cloning, tissue expression pattern, and chromosomal mapping.
AU Hosokawa, Yoshitaka [Reprint author]; Maeda, Yumiko; Takahashi, Ei-ichi; Suzuki, Mikio; Seto, Masao
CS Laboratory of Chemotherapy, Aichi Cancer Center Research Institute, 1-1 Kanakoden, Chikusa-ku, Nagoya, 464-8681, Japan
SO Genomics, (Nov. 1, 1999) Vol. 61, No. 3, pp. 326-329. print.
CODEN: GNMCEP. ISSN: 0888-7543.
DT Article
LA English
ED Entered STN: 9 Feb 2000
Last Updated on STN: 3 Jan 2002
AB The ***Ikaros*** gene (symbol ZNFN1A1) encodes the hematopoietic zinc finger DNA binding protein, which is now recognized as a central regulator of lymphoid differentiation and has been implicated in leukemogenesis. Recently, an ***Ikaros*** -related zinc finger protein, called Aiolos (ZNFN1A3), has been identified and characterized, thus establishing the presence of a gene family whose members may be hematopoietic transcription factors. Among Aiolos-mutant mice, development of B-cell lymphoma was frequently seen. As an initial approach to examining the possible involvement of Aiolos in the pathogenesis of human lymphoid proliferative disease, we isolated cDNA clones for human Aiolos from a B-cell cDNA library. The human Aiolos protein predicted from the cDNA sequence consists of 509 amino acid residues and shares 86% sequence identity with its mouse counterpart. As in the case with mouse Aiolos, no isoform for human Aiolos has been found. Northern blot analysis of various human tissues revealed that the Aiolos transcripts are expressed most strongly in peripheral blood leukocytes, the spleen, and the thymus, supporting the notion that Aiolos plays an important role in lymphoid lineages. Fluorescence in situ hybridization using a ***BAC*** clone established that the Aiolos gene is mapped to human chromosome band 17q11.2.

L6 ANSWER 8 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2000:27273 BIOSIS
DN PREV200000027273
TI Isolation and characterization of a novel TP53-inducible gene, TP53TG3.
AU Ng, Ching Ching; Koyama, Kumiko; Okamura, Shu; Kondoh, Hisato; Takei, Yoshiki; Nakamura, Yusuke [Reprint author]
CS Laboratory of Molecular Medicine, Human Genome Center, Institute of Medical Science, University of Tokyo, 4-6-1, Shirokanedai, Minato-ku, Tokyo, 108-8639, Japan
SO Genes Chromosomes and Cancer, (Dec., 1999) Vol. 26, No. 4, pp. 329-335. print.
CODEN: GCCAES. ISSN: 1045-2257.
DT Article
LA English
ED Entered STN: 13 Jan 2000
Last Updated on STN: 31 Dec 2001
AB We applied the differential mRNA display method to isolate genes regulated by wild-type TP53 in cells of a colon-cancer line (SW480) in which we had established an inducible TP53 expression system under the control of the lactose operon. Here we report isolation and characterization of a novel TP53-inducible gene, termed TP53TG3 (TP53 target gene 3). Its DNA sequence was identical to sequences present in two ***BAC*** clones that had been mapped to chromosome band 16p13. The gene expressed several
transcripts by alternative splicing; the two major transcripts, TP53TG3a and TP53TG3b, encoded 124- and 132-amino-acid peptides that were expressed
predominantly in testis. Immunohistochemical analysis using cancer cells (HeLa or H1299) that had been transfected with plasmid DNA designed to express the ***MYC*** -fused TP53TG3 proteins indicated that these products were present mainly in the cytoplasm 20 hr after transfection. However, 40 hr after transfection, the recombinant proteins had accumulated inthe nuclei of some cells. Because no known nuclear localization domain was present in the amino acid sequence, we suspect that this protein plays an important role in the TP53-mediated signaling pathway, when it forms complexes with other protein(s) and is transferred by them into the nucleus.

L6 ANSWER 9 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2000:25549 BIOSIS
DN PREV200000025549
TI B-cell tumorigenesis in mice carrying a yeast ***artificial*** ***chromosome*** -based immunoglobulin heavy/c- ***myc*** translocus is independent of the heavy chain intron enhancer (Emu).
AU Palomo, Concepcion; Zou, Xiangang; Nicholson, Ian C.; Butzler, Christoph;

Bruggemann, Marianne [Reprint author]
CS Laboratory of Developmental Immunology, Department of Development and Genetics, Babraham Institute, Babraham, Cambridge, CB2 4AT, UK
SO Cancer Research, (Nov. 1, 1999) Vol. 59, No. 21, pp. 5625-5628. print.
CODEN: CNREA8. ISSN: 0008-5472.
DT Article
LA English
ED Entered STN: 29 Dec 1999
Last Updated on STN: 31 Dec 2001
AB We have used ***YAC*** (yeast ***artificial*** ***chromosome***) technology to create large translocation regions where the c- ***myc*** proto-oncogene is coupled to the core region of the human immunoglobulin heavy chain (IgH) locus (from VH2-5 through to Cdelta). Chimeric mice were obtained from embryonic stem cells carrying a single copy of the 240-kb IgH/c- ***myc*** translocation region. B-cell tumorigenesis occurs in the translocus mice, even when the entire Emu intron enhancer region between the joining segments and switch mu is deleted. This demonstrates that as yet unidentified regulatory elements in the IgH locus, independent from the known enhancers, are sufficient to cause B-cell specific activation of c- ***myc*** after translocation. The phenotype of tumors from IgH/c- ***myc*** ***YAC*** transgenic mice with or without Emu (B220+, IgM+/IgD+) is reminiscent of Burkitt's lymphoma. A rapidly expanding abnormal B-cell population is present at birth and accumulates in bone marrow, periphery, and spleen, well before discrete tu mor establishment. Molecular analysis identified a clonal origin, with rearrangement of one mouse heavy chain allele retained in tumor cells from different sites, whereas subsequent rearrangements of heavy or light chain loci can be diverse. These mice routinely develop mature B-cell tumors early in life and may provide an invaluable resource of a B-cell lymphoma model.

L6 ANSWER 10 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1999:8411 BIOSIS
DN PREV199900008411
TI Subtracted, unique-sequence, in situ hybridization: Experimental and diagnostic applications.
AU Davison, Jon M.; Morgan, Thomas W.; Hsi, Bae-Li; Xiao, Sheng; Fletcher, Jonathan A. [Reprint author]
CS Dep. Pathol., Brigham and Women's Hosp., 75 Francis St., Boston, MA 02115,
USA
SO American Journal of Pathology, (Nov., 1998) Vol. 153, No. 5, pp. 1401-1409. print.
CODEN: AJPAA4. ISSN: 0002-9440.
DT Article
LA English
ED Entered STN: 11 Jan 1999
Last Updated on STN: 11 Jan 1999
AB Nonrandom chromosomal aberrations, particularly in cancer, identify pathogenic biological pathways and, in some cases, have clinical relevance as diagnostic or prognostic markers. Fluorescence and colorimetric in situ hybridization methods facilitate identification of numerical and structural chromosome abnormalities. We report the development of robust, unique-sequence in situ hybridization probes that have several novel features: 1) they are constructed from multimegabase contigs of yeast ***artificial*** ***chromosome*** (***YAC***) clones; 2) they are in the form of adapter-ligated, short-fragment, DNA libraries that may be amplified by polymerase chain reaction, and 3) they have had repetitive sequences (eg, Alu and LINE elements) quantitatively removed by subtractive hybridization. These subtracted probes are labeled conveniently, and the fluorescence or colorimetric detection signals are extremely bright. Moreover, they constitute a stable resource that may be amplified through at least four rounds of polymerase chain reaction without diminishing signal intensity. We demonstrate applications of subtracted probes for the ***MYC*** and EWS oncogene regions, including 1) characterization of a novel EWS-region translocation in Ewing's sarcoma, 2) identification of chromosomal translocations in paraffin sections, and 3) identification of chromosomal translocations by conventional bright-field microscopy.

=> d bib abs 11-20

L6 ANSWER 11 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:498132 BIOSIS
DN PREV199800498132
TI Simultaneous detection of ***MYC*** , BVRI, and PVTI translocations in lymphoid malignancies by fluorescence in situ hybridization.
AU Rack, Katrina A.; Delabesse, Eric; Radford-Weiss, Isabelle; Bourquelot, Priscille; Le Guyader, Gaelle; Vekemans, Michel; Macintyre, Elizabeth A. [Reprint author]
CS Lab. d'Hematol., Tour Pasteur, Hopital Necker-Enfants Malades, 149 rue des Sevres, 75743 Paris Cedex 15, France
SO Genes Chromosomes and Cancer, (Nov., 1998) Vol. 23, No. 3, pp. 220-226. print.
CODEN: GCCAES. ISSN: 1045-2257.
DT Article
LA English
ED Entered STN: 18 Nov 1998
Last Updated on STN: 18 Nov 1998
AB The rapid detection of chromosome band 8q24 rearrangements, including classical translocations involving ***MYC*** and variant 3' translocations, is important for the accurate diagnosis and appropriate treatment of lymphoid malignancies. We have identified and characterized a CEPH ***YAC*** , 934el, which extends from at least 190 kbp upstream to over 280 kbp downstream to M YC, allowing detection of classical t(8;14)(q24;q32) and variant t(8;22)(q24;q11) and t(8;14)(q24;q11), extending distal to PVTI and therefore, by extrapolation, to BVRI. This ***YAC*** also allowed clarification of complex chromosome 8 abnormalities and the identification of translocations in interphase nuclei. A second CEPH ***YAC*** , 904c3, previously shown to contain the PVTI locus but not ***MYC*** , allowed distinction between translocations occurring centromeric and telomeric to ***MYC*** . Use of the 934el ***YAC*** will aid classification of a variety of lymphoid proliferations and further characterization of rearranged cases with the 904c3 ***YAC*** will simplify mapping of their diverse breakpoints.

L6 ANSWER 12 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:364836 BIOSIS
DN PREV199800364836
TI A variant Burkitt-type translocation (8;22)(q24;q11) in multiple myeloma: Report of a new case and review of the literature.
AU Yamamoto, Katsuya; Hamaguchi, Hiroyuki [Reprint author]; Nagata, Kaoru; Taniwaki, Masafumi
CS Dep. Hematol., Musashino Red Cross Hosp., 1-26-1 Kyonan-cho, Musashino, Tokyo 180, Japan
SO Cancer Genetics and Cytogenetics, (July 15, 1998) Vol. 104, No. 2, pp. 98-103. print.
CODEN: CGCYDF. ISSN: 0165-4608.
DT Article
LA English
ED Entered STN: 27 Aug 1998
Last Updated on STN: 27 Aug 1998
AB We report here a new case of multiple myeloma (IgG, kappa, stage IIIA) with a variant Burkitt-type translocation (8,22)(q24;q11). Bone marrow plasma cells were morphologically immature with fine nuclear chromatin and nucleoli. Chromosome analysis showed complex aberrations; that is, 53,XY, der(1)add(1)(p11)dup(1)(q12q32),+3,+5, t(8;22)(q24;q11), +9, add(10)(p13),+11, +15,add(19)(q13),+21. Fluorescence in situ hybridization analysis with the yeast ***artificial*** ***chromosome*** (***YAC***) clone 12 containing the C- ***MYC*** gene at 8q24 and the chromosome-22-specific DNA library pBS22 revealed that 12 was located on the der(8)t(8;22). A fusion signal derived from l2 and the ***YAC*** clone B99E1 containing the BCR gene at 22q11 was also observed on the der(8)t(8;22). Our results indicate that the breakpoint at 8q24 in this patient was located far downstream of the C- ***MYC*** gene. This breakpoint site is similar to Burkitt lymphoma with t(8;22)(q24;q11). A review of eight cases in the literature and the present case of multiple myeloma with t(8;22)(q24;q11) showed that most of them were of advanced stage and had an immature phenotype. It is suggested that the C- ***MYC*** gene may be activated by t(8,22)(q24;q11) and implicated in disease progression in multiple myeloma.

L6 ANSWER 13 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:361904 BIOSIS
DN PREV199800361904
TI Genomic organization and chromosomal localization of the murine epididymal retinoic acid-binding protein (mE-RABP) gene.
AU Lareyre, Jean-Jacques; Mattei, Marie-Genevieve; Kasper, Susan; Ong, David E.; Matusik, Robert J.; Orgebin-Crist, Marie-Claire [Reprint author]
CS Cent. Reproductive Biol. Res., Vanderbilt Univ., Sch. Med., Medical Center N., Room D2303, Nashville, TN 37233-2633, USA
SO Molecular Reproduction and Development, (Aug., 1998) Vol. 50, No. 4, pp. 387-395. print.
CODEN: MREDEE. ISSN: 1040-452X.
DT Article
LA English
ED Entered STN: 27 Aug 1998
Last Updated on STN: 27 Aug 1998
AB The murine epididymal retinoic acid-binding protein (mE-RABP) is specifically synthesized in the mouse mid/distal caput epididymidis and secreted in the lumen. In this report, we have demonstrated by Southern blot analysis of genomic DNA that mE-RABP is encoded by a single-copy gene. A mouse 129/SvJ genomic bacteria) ***artificial*** ***chromosome*** (***BAC***) library was screened using a cDNA encoding the minor form of mE-RABP. One positive ***BAC*** clone was characterized and sequenced to determine the nucleotide sequence of the entire mE-RABP gene. The molecular cloning of the mE-RABP gene completes the characterization of the 20.5-kDa-predicted preprotein leading to the minor and major forms of ME-RABP. Comparison of the DNA sequence of the promoter and coding regions with that of the rat epididymal secretory protein I (ESP 1) gene showed that the mE-RABP gene is the orthologue of the ESPI gene that encodes a rat epididymal retinoic acid-binding, protein. Several regulatory elements, including a putative androgen receptor binding site, "CACCC-boxes," NF-1, ***Oct*** - ***1*** , and SP-1 recognition sites, are conserved in the proximal promoter. Analysis of the nucleotde sequence of the mE-RABP gene-revealed the presence of seven exons and showed that the genomic organization is highly related to other genes encoding lipocalins. The mE-RABP gene was mapped by fluorescent in situ hybridization to the (A3-B) region of the murine

chromosome 2. Our data, combined with that of others, suggest that the proximal segment of the mouse chromosome 2 may be a rich region for genes encoding lipocalins with a genomic organization highly related to the mE-RABP gene.

L6 ANSWER 14 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:227957 BIOSIS
DN PREV199800227957
TI Rapid isolation of cDNA by hybridization.
AU Hamaguchi, Masaaki; O'Connor, Elizabeth A.; Chen, Tong; Parnell, Larry; McCombie, Richard W.; Wigler, Michael H. [Reprint author]
CS Cold Spring Harbor Lab., 1 Bungtown Road, Cold Spring Harbor, NY 11724, USA
SO Proceedings of the National Academy of Sciences of the United States of America, (March 31, 1998) Vol. 95, No. 7, pp. 3764-3769. print.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 20 May 1998
Last Updated on STN: 20 May 1998
AB The isolation of genes from a given genomic region can be a rate-limiting step in the discovery of disease genes. We describe an approach to the isolation of cDNAs that have sequences in common with large genomic clones such as bacterial ***artificial*** ***chromosomes*** . We applied this method to loci both amplified and deleted in cancer, illustrating its usage in the identification of both oncogenes and tumor suppressor genes, respectively. The method, called rapid isolation of cDNAs by hybridization (RICH), depends on solution hybridization, enzymatic modification, and amplification/selection of sequences present in both cDNA populations and the genomic clones. The method should facilitate the development of transcription maps for large genomic clones, possibly even yeast ***artificial*** ***chromosomes*** .

L6 ANSWER 15 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:121286 BIOSIS
DN PREV199800121286
TI A distamycin A-inducible fragile site, FRA8E, located in the region of the hereditary multiple exostoses gene, is not involved in HPV 16 DNA integration and amplification.
AU Hori, Tada-Aki [Reprint author]; Seki, Naohiko; Ohira, Miki; Saito, Toshiyuki; Yamauchi, Masatake; Sagara, Masahi; Hayashi, Akiko; Tsuji, Satsuki; Ito, Hiroko; Imai, Takashi
CS Genome Res. Group, Natl. Inst. Radiological Sci., 4-9-1 Anagawa, Inage-ku, Chiba 263, Japan
SO Cancer Genetics and Cytogenetics, (Feb., 1998) Vol. 101, No. 1, pp. 24-34. print.
CODEN: CGCYDF. ISSN: 0165-4608.
DT Article
LA English
ED Entered STN: 5 Mar 1998
Last Updated on STN: 5 Mar 1998
AB The rare fragile site is a specific point on a chromosome that is expressed as an isochromatid gap or break under certain conditions of cell culture and is inherited in a Mendelian codominant fashion. Five folate-sensitive fragile sites were cloned, and the molecular basis of fragile site mutation was shown to be a new class of mutation, called dynamic mutation, resulting from an allelic expansion of (CCG)n repeats. The mechanism responsible for other types of rare fragile sites, i.e., distamycin A-inducible and BrdU-requiring, is unknown, although cytogenetic studies suggested that these fragile sites play a mechanistic role in breakage and recombination and may also be integration and modification sites of foreign viral DNA genomes. A distamycin A-inducible fragile site, FRA8E, is mapped to 8q24.1 in which various loci implicated in genomic instability are located. Here we identified a ***YAC*** clone spanning both FRA8E and the hereditary multiple exostosis (EXT1) gene, using fluorescence in situ hybridization (FISH) analysis of a yeast ***artificial*** ***chromosome*** (***YAC***) contig. By using P1 clones as probes, the FRA8E locus was further localized to a 400-kb region including the EXT1 gene. Furthermore, the integration and amplification site of human papillomavirus 16 DNA in the ASCC (argyrophil small cell carcinoma) cells were shown not to coincide with FRA8E, but to be involved in an extensively broad genomic region of 8q24.1, including the c- ***myc*** gene.

L6 ANSWER 16 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1997:213826 BIOSIS
DN PREV199799520330
TI Rapid induction of B-cell lymphomas in mice carrying a human IgH/c-mycYAC.
AU Butzler, Christoph; Zou, Xiangang; Popov, Andrei V.; Bruggemann, Marianne
CS Dep. Development Genetics, Babraham Inst., Babraham, Cambridge CB2 4AT, UK
SO Oncogene, (1997) Vol. 14, No. 11, pp. 1383-1388.
CODEN: ONCNES. ISSN: 0950-9232.
DT Article
LA English
ED Entered STN: 22 May 1997
Last Updated on STN: 22 May 1997
AB Activation of the c- ***myc*** proto-oncogene by one of the immunoglobulin (Ig) loci after chromosomal translocation is a consistent feature of Burkitt's lymphoma. Different subtypes of this tumor vary in the molecular architecture of the translocation region. In most cases there are no known regulatory elements of the Ig locus neighboring the oncogene and this considerably obscures the mechanism of its deregulation. In order to assess possible oncogene activation signals, we produced an experimental translocation region by insertion of a c- ***myc*** gene about 50 kb from the IgH intron enhancer in a yeast ***artificial*** ***chromosome*** (***YAC***) containing a 220 kb region of the human Ig heavy chain (IgH) locus. Single copy integration of this ***YAC*** into the genome of mouse embryonic stem (ES) cells was achieved by spheroplast fusion. Chimeric mice derived from these ES cells developed monoclonal B-cell lymphomas expressing surface IgM by 8-16 weeks of age. The IgH/c- ***myc*** translocus showed different V-HDJ-H rearrangement in almost all tumors without any alterations of the distance between c- ***myc*** and the IgH intron enhancer. This mouse model can be used for the in vivo analysis of c- ***myc*** deregulation and the tumor formation capacity of the IgH locus in aberrant rearrangements.

L6 ANSWER 17 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1996:364326 BIOSIS
DN PREV199699086682
TI Genomic structure and precise mapping of a thymic regulatory region on mouse chromosome 17 revealed by a c- ***myc*** transgene insertion.
AU Lavenu, A. [Reprint author]; Roland, J.; Poirier, C.; Cazenave, P.-A.; Babinet, C.; Morello, D.
CS Dep. Immunol., Unite de Biologie du Developpement CNRS URA 1960, Inst. Pasteur, 25 rue du Dr. Roux, 75724 Paris, France
SO Genomics, (1996) Vol. 34, No. 3, pp. 381-388.
CODEN: GNMCEP. ISSN: 0888-7543.
DT Article
LA English
ED Entered STN: 14 Aug 1996
Last Updated on STN: 14 Aug 1996
AB In one transgenic strain harboring a human c- ***myc*** proto-oncogene construct, the transgene was actively and exclusively expressed in the thymus, where it contributed to the development of lymphoma that corresponded to CD4+CD8+ cells. Here, we have pursued the analysis of transgene expression in healthy transgenic mice and show that transgene activation occurs in the thymus 3 days before birth, at a time when CD4+CD8+ lymphocytes emerge. In the adult, its expression is restricted to the CD4+CD8+ cells. The region flanking the transgene insertion site was isolated and made it possible to map the preintegration locus, hereafter called Tsil (for thymus-specific integration locus) on chromosome 17 between D17Rp11e and Ras12-3. A ***YAC*** that contains both Tsil and the Pim2 locus, previously shown to be involved in progression of T-cell lymphoma, was isolated. Analysis of Tsil offers a unique opportunity to identify a regulatory region or a gene that might play an important role in T-cell maturation.

L6 ANSWER 18 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1995:255242 BIOSIS
DN PREV199598269542
TI Detection of ***myc*** translocations in lymphoma cells by fluorescence in situ hybridization with yeast ***artificial*** ***chromosomes*** .
AU Veronese, Maria Luisa [Reprint author]; Ohta, Masataka; Finan, Janet; Nowell, Peter C.; Croce, Carlo M.
CS Jefferson Cancer Inst., Jefferson Med. Coll., BLBS, Room 1032, 233 S. 10th St., Philadelphia, PA 19107, USA
SO Blood, (1995) Vol. 85, No. 8, pp. 2132-2138.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Article
LA English
ED Entered STN: 13 Jun 1995
Last Updated on STN: 13 Jun 1995
AB Translocations involving chromosome 8 at band q24 and one of the Ig loci on chromosomes 14q32, 22q11, and 2p11 are the hallmark of Burkitt's lymphoma (BL). It has been previously observed that the exact localization of the breakpoints at chromosome 8q24 can vary significantly from patient to patient, scattering over a distance of more than 300 kb upstream of c- ***myc*** and about 300 kb downstream of c- ***myc*** . To generate probes for fluorescence in situ hybridization (FISH) that detect most c- ***myc*** translocations, we screened a yeast ***artificial*** ***chromosome*** (***YAC***) library from normal human lymphocytes by colony hybridization, using three markers surrounding the c- ***myc*** gene as probes. We obtained 10 ***YAC*** clones ranging in size between 500 and 200 kb. Two nonchimeric clones were used for FISH on several BL cell lines and patient samples with different breakpoints at 8q24. Our results show that the ***YAC*** clones detected translocations scattered along approximately 200 kb in both metaphase chromosomes and interphase nuclei. The sensitivity, rapidity, and feasibility in nondividing cells render FISH an important diagnostic tool. Furthermore, the use of large DNA fragments such as YACs greatly simplifies the detection of translocations with widely scattered breakpoints such as these seen in BL.

L6 ANSWER 19 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1995:171870 BIOSIS
DN PREV199598186170
TI Identification of ***YAC*** clones for human chromosome 1p32 and physical mapping of the infantile neuronal ceroid lipofuscinosis (INCL)

locus.
AU Hellsten, E.; Vesa, J.; Heiskanen, M.; Makela, T. P.; Jarvela, I.; Cowell, J. K.; Mead, S.; Alitalo, K.; Palotie, A.; Peltonen, L. [Reprint author]
CS Dep. Human Molecular Genetics, National Public Health Inst., Mannerheimintie 166, 00300 Helsinki, Finland
SO Genomics, (1995) Vol. 25, No. 2, pp. 404-412.
CODEN: GNMCEP. ISSN: 0888-7543.
DT Article
LA English
ED Entered STN: 26 Apr 1995
Last Updated on STN: 26 Apr 1995
AB Infantile neuronal ceroid lipofuscinosis (INCL, CLN1) is a neurodegenerative disorder in which the biochemical defect is unknown. We earlier assigned the disease locus to chromosome 1p32 in the immediate vicinity of the highly informative HY-TM1 marker by linkage and linkage disequilibrium analysis. Here we report the construction of PFGE maps on the CLN1 region covering a total of 4 Mb of this relatively poorly mapped chromosomal region. We established the order of loci at 1p32 as tel-D1S57-L- ***myc*** -HY-TM1-rlf-COL9A2-D1S193-D1S62-D1S211-cen by combining data obtained from analysis of a chromosome 1 somatic cell hybrid panel, PFGE, and interphase FISH. We isolated YACs and constructed two separate ***YAC*** contigs, the loci L- ***myc*** , HY-TM1, rlf, and COL9A2 being present on a 1000-kb contig and the markers D1S193, D1S62, and D1S211 on a ***YAC*** contig spanning a maximum of 860 kb. Within the 1000-kb contig we were able to identify five CpG islands in addition to those associated with the earlier cloned genes. The ***YAC*** contigs as well as the physical map provide us with tools for the identification of the INCL gene.

L6 ANSWER 20 OF 45 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1995:171277 BIOSIS
DN PREV199598185577
TI The gene encoding the VP16-accessory protein HCF (HCFC1) resides in human
Xq28 and is highly expressed in fetal tissues and the adult kidney.
AU Wilson, Angus C.; Parrish, Julia E.; Massa, Hillary F.; Nelson, David L.; Trask, Barbara J.; Herr, Winship [Reprint author]
CS Cold Spring Harbor Lab., P.O. Box 100, Cold Spring Harbor, NY 11724, USA
SO Genomics, (1995) Vol. 25, No. 2, pp. 462-468.
CODEN: GNMCEP. ISSN: 0888-7543.
DT Article
LA English
ED Entered STN: 26 Apr 1995
Last Updated on STN: 26 Apr 1995
AB After herpes simplex virus (HSV) infection, the viral regulatory protein VP16 activates transcription of the HSV immediate-early promoters by directing complex formation with two cellular proteins, the POU-homeodomain transcription factor ***Oct*** - ***1*** and the host cell factor HCF. The function of HCF in uninfected cells is unknown. Here we show by fluorescence in situ hybridization and somatic cell hybrid analysis that the gene encoding human HCF, HCFC1, maps to the q28 region of the X chromosome. Yeast ***artificial*** ***chromosome*** and cosmid mapping localizes the HCFC1 gene within 100 kb distal of the renal vasopressin type-2 receptor (V2R) gene and adjacent to the renin-binding protein gene (RENBP). The HCFC1 gene is apparently unique. HCF transcripts and protein are most abundant in fetal and placental tissues and cell lines, suggesting a role in cell proliferation. In adults, HCF protein is abundant in the kidney, but not in the brain, a site of latent HSV infection and where HCF levels may influence progression of HSV infection.

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NEWS 10 DEC 08 CABA reloaded with left truncation
NEWS 11 DEC 08 IMS file names changed
NEWS 12 DEC 09 Experimental property data collected by CAS now available in REGISTRY
NEWS 13 DEC 09 STN Entry Date available for display in REGISTRY and CA/CAplus
NEWS 14 DEC 17 DGENE: Two new display fields added
NEWS 15 DEC 18 BIOTECHNO no longer updated
NEWS 16 DEC 19 CROPU no longer updated; subscriber discount no longer available
NEWS 17 DEC 22 Additional INPI reactions and pre-1907 documents added to CAS databases
NEWS 18 DEC 22 IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields
NEWS 19 DEC 22 ABI-INFORM now available on STN
NEWS 20 JAN 27 Source of Registration (SR) information in REGISTRY updated and searchable
NEWS 21 JAN 27 A new search aid, the Company Name Thesaurus, available in CA/CAplus
NEWS 22 FEB 05 German (DE) application and patent publication number format changes
NEWS 23 MAR 03 MEDLINE and LMEDLINE reloaded
NEWS 24 MAR 03 MEDLINE file segment of TOXCENTER reloaded
NEWS 25 MAR 03 FRANCEPAT now available on STN

NEWS EXPRESS MARCH 5 CURRENT WINDOWS VERSION IS V7.00A, CURRENT
MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
AND CURRENT DISCOVER FILE IS DATED 3 MARCH 2004
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FULL ESTIMATED COST 0.21 0.21

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=> s artificial chromosom? or PAC or BAC or YAC
L1 35029 ARTIFICIAL CHROMOSOM? OR PAC OR BAC OR YAC

=> s l1 and Ikaros
L2 15 L1 AND IKAROS

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 9 DUP REM L2 (6 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 9 ANSWERS - CONTINUE? Y/(N):y

L3 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 1
AN 2003:187091 CAPLUS
DN 138:219713

TI Differentially expressed gene expression profiles in human glomerular diseases
IN Munger, William E.; Falk, Ronald; Sun, Hongwei; Sasai, Hitoshi; Waga, Iwao; Yamamoto, Jun
PA Gene Logic, Inc., USA; University of North Carolina At Chapel Hill
SO PCT Int. Appl., 781 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 9

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003016476	A2	20030227	WO 2002-XH25766	20020814
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2003016476	A2	20030227	WO 2002-US25766	20020814
WO 2003016476	A3	20030508		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRAI US 2001-311837P P 20010814
WO 2002-US25766 A 20020814
AB The present invention is based on the elucidation of global changes in gene expression in peripheral blood leukocytes (PBL) of patients with glomerular diseases exhibiting different types of clin. and pathol. features of glomerular nephropathy as compared to normal PBL as well as the identification of individual genes that are differently expressed in PBL of patients with glomerular diseases. The genes and gene expression information may be used as markers for the diagnosis of disease subtype, such as IgA nephropathy, Minimal Change nephrotic syndrome, antineutrophil cytoplasmic antibody-assocd. glomerulonephritis (ANCA), focal segmental glomerulosclerosis (FSGS), and lupus nephritis. The genes may also be used as markers to evaluate the effects of a candidate drug or agent on tissues, including PBLs, particularly PBLs undergoing activation or PBLs from a patient with glomerular disease. Differential expression of genes between PBLs from patients with glomerular disease and normal PBL samples was detd. using the Affymetrix 42K human gene chip set. [This abstr. record is one of nine records for this document necessitated by the large no. of index entries required to fully index the document and publication system constraints.].

L3 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:913280 CAPLUS
DN 139:379453
TI Genes showing altered patterns of expression in multiple sclerosis and their diagnostic and therapeutic uses
IN Dangond, Fernando; Hwang, Daehee
PA Brigham and Women's Hospital, Inc., USA
SO PCT Int. Appl., 148 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003095618	A2	20031120	WO 2003-US14462	20030507
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004018522	A1	20040129	US 2003-430762	20030506

PRAI US 2002-379284P P 20020509
US 2003-430762 A1 20030506
AB The present invention identifies a no. of gene markers whose expression is altered in multiple sclerosis (MS). These markers can be used to diagnose or predict MS in subjects, and can be used in the monitoring of therapies. In addn., these genes identify therapeutic targets, the modification of which may prevent MS development or progression. Genes were identified by detn. of expression profiling. A large no. of genes showing altered patterns of expression were identified, with the most discriminatory genes being those for: phosphatidylinositol transfer protein, inducible nitric oxide synthase, ClC-1 (CLCN1) muscle chloride channel protein, placental bikunin (AMBP), receptor kinase ligand LERK-3/Ephrin-A3, GATA-4, thymopoietin, transcription factor E2f-2, S-adenosylmethionine synthetase, carcinoembryonic antigen, the ret oncogene, a G protein-linked receptor (clone GPCR W), GTP- binding protein RALB, tyrosine kinase Syk, LERK-2/Ephrin-B1, ELK1 tyrosine kinase oncogene, transcription factor SL1, phospholipase C, gastricsin (progastricsin), and the D13S824E locus.

L3 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:875393 CAPLUS
DN 139:363045
TI Genes expressed in atherosclerotic tissue and their use in diagnosis and pharmacogenetics
IN Nevins, Joseph; West, Mike; Goldschmidt, Pascal
PA Duke University, USA
SO PCT Int. Appl., 408 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003091391	A2	20031106	WO 2002-US38221	20021112
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2003091391	A2	20031106	WO 2002-XA38221	20021112
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
WO 2003091391	A2	20031106	WO 2002-XB38221	20021112
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003224383	A1	20031204	US 2002-291885	20021112

PRAI US 2002-374547P P 20020423
US 2002-420784P P 20021024
US 2002-421043P P 20021025
US 2002-424680P P 20021108
WO 2002-US38221 A 20021112
AB Genes whose expression is correlated with an determinant of an atherosclerotic phenotype are provided. Also provided are methods of using the subject atherosclerotic determinant genes in diagnosis and treatment methods, as well as drug screening methods. In addn., reagents and kits thereof that find use in practicing the subject methods are provided. Also provided are methods of detg. whether a gene is correlated with a disease phenotype, where correlation is detd. using a Bayesian anal.

L3 ANSWER 4 OF 9 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:475871 BIOSIS
DN PREV200300475871
TI High-resolution analysis of chromosomal imbalances in mouse lymphomas using whole genome ***BAC*** arrays.
AU Cai, Wei-Wen [Reprint Author]; Mao, Jian-Hua; Li, Jianzhen; Jiang, Tao; Balmain, Allan; Justice, Monica; Peterson, Leaf
CS Department of Molecular and Human Genetics, Baylor College of Medicine, Houston, TX, USA
SO Proceedings of the American Association for Cancer Research Annual Meeting, (July 2003) Vol. 44, pp. 957. print.
Meeting Info.: 94th Annual Meeting of the American Association for Cancer Research. Washington, DC, USA. July 11-14, 2003.
ISSN: 0197-016X.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 15 Oct 2003
Last Updated on STN: 15 Oct 2003

L3 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:716967 CAPLUS
DN 137:231381
TI Immunoglobulin control region
IN Mundt, Cornelia Anna; Brueggemann, Marianne
PA UK
SO U.S. Pat. Appl. Publ., 21 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2002132373	A1	20020919	US 2002-81599	20020220
PRAI GB 2001-4299	A	20010221		

AB The region between C.delta. and C.gamma.3 of IgH is unstable and may be a recombination hot spot. It has now been shown that 21 kb of the unstable region in the human IgH locus between C.delta. and C.gamma.3 contains a highly clustered array of a large no. of transcription factor-binding motifs interspersed with repeat sequences. Transfection assays revealed transcription enhancement and silencing activity at the pre B-cell stage and in transgenic mice strong enhancer function was identified in the bone marrow, the primary site of B-cell differentiation. Flow cytometry anal. of early B-cell populations showed that this enhancer is already active at the pro/pre B-cell stage where DNA rearrangement is initiated. The region accommodating E.delta.-.gamma.3 may exert locus control function at an early developmental stage, which may be crit. in normal and aberrant B-cell development.

L3 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:783406 CAPLUS
DN 138:364779
TI Genetic analysis of radiation-induced thymic lymphoma
AU Kominami, R.; Saito, Y.; Shinbo, T.; Matsuki, A.; Kosugi-Okano, H.; Matsuki, A.; Ochiai, Y.; Kodama, Y.; Wakabayashi, Y.; Takahashi, Y.; Mishima, Y.; Niwa, O.
CS Department of Gene Regulation, Niigata University Graduate School of Medical and Dental Sciences, Niigata, 951-8122, Japan
SO International Congress Series (2002), 1236(Radiation and Homeostasis), 143-150
CODEN: EXMDA4; ISSN: 0531-5131
PB Elsevier Science B.V.
DT Journal
LA English
AB Mouse thymic lymphomas are one of the classic models of radiation-induced malignancies. However, little genetic study has been performed, although the mouse systems offer a no. of useful features for genetic and phys. mapping. We have carried out large-scale mapping toward the isolation of the genes involved in lymphoma development. Two different types of genes are chosen as targets for positional cloning. One is the tumor suppressor gene and the other is the susceptibility or resistance-giving gene, which predispose to the lymphoma development. One susceptibility locus was localized near D4Mit12 on chromosome 12 by an assocn. study for backcross and congenic mice, and three loci, probably harboring a tumor suppressor gene, were localized by allelic loss mapping on phys. maps that were covered by ***BAC*** clones. The maps are invaluable to facilitate the identification of candidate tumor suppressor genes. Also, success in identification of ***Ikaros*** as a tumor suppressor gene is described.
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 7 OF 9 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 2
AN 2001:409592 BIOSIS
DN PREV200100409592
TI Genomic organization and promoter analysis of human KCNN3 gene.
AU Sun, Guifeng; Tomita, Hiro-aki; Shakkottai, Vikram G.; Gargus, J. Jay [Reprint author]
CS Department of Physiology and Biophysics, University of California, Irvine, Med Sci I D-335, Irvine, CA, 92697-4560, USA
JJGARGUS@UCI.edu
SO Journal of Human Genetics, (2001) Vol. 46, No. 8, pp. 463-470. print.
ISSN: 1434-5161.
DT Article
LA English
ED Entered STN: 29 Aug 2001
Last Updated on STN: 22 Feb 2002
AB KCNN3 is a member of the gene family, KCNN1-4, encoding the small and intermediate conductance calcium-activated potassium channels. Long CAG-repeat alleles of this gene have been found to be over-represented in patients with schizophrenia in a number of population-based association studies, and this gene maps to human chromosome 1q21, a region recently implicated in schizophrenia by linkage. To set the stage for a further functional evaluation of KCNN3, we defined the nature of the genomic locus in the size, structure, and sequence of its introns and exons and the function of potential upstream regulatory regions. We isolated P1-derived ***artificial*** ***chromosome*** (***PAC***) clones from a genomic library and identified an overlapping available bacterial ***artificial*** ***chromosome*** (***BAC***) clone. Cosmids subcloned from the ***PAC*** and ***BAC*** clones were then sequenced and merged with the sequence in the public database. The KCNN3 gene spans over 163.1 kb and is composed of eight exons and seven introns. All of the exon-intron junctions conform closely to consensus splice sites. The proximal 2.5 kb of the 5'-flanking sequence was obtained and analyzed for potential transcription factor binding sites. In the proximal 2.5 kb upstream region, potential sites for the ***Ikaros*** factor (IK2), homeodomain factor Nkx-2.5/Csx (NKX25), nuclear factor of activated T-cells (NFAT), upstream stimulating factor (USF), c-AMP responsive element binding protein (CREB), POU factor Brn2 (BRN-2), myeloid zinc finger protein (MZF1), vitellogenin binding protein (VBP), HNF3 forkhead homologue 2 (HFH2), and transcription initiation were identified, as well as several potential AP-1 and AP-4 sites. Finally, a 2261-bp fragment of this upstream region was cloned into a promoterless pGL3-luciferase vector, where it produced orientation-dependent expression of the reporter gene in transiently transfected PC12 cells, cells which natively express functional KCNN3 channels, suggesting that this cloned fragment includes competent promoter elements of this gene.

L3 ANSWER 8 OF 9 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:315001 BIOSIS
DN PREV200100315001
TI A genetic screen in the zebrafish defines mutations required for the development of the immune system.
AU Trede, Nikolaus S. [Reprint author]; Ota, Tatsuya; Kawasaki, Hirohide; Barut, Bruce [Reprint author]; Paw, Barry H. [Reprint author]; Zhou, Yi [Reprint author]; Hersey, Candance [Reprint author]; Zapata, Augustin; Amemiya, Chris T.; Zon, Leonard I. [Reprint author]
CS Div. Hematology, Children's Hospital, Boston, MA, USA
SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 613a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 4 Jul 2001
Last Updated on STN: 19 Feb 2002
AB Few genes have been identified that regulate early lymphoid and thymic development. In all vertebrates studied to date, the thymus buds off the endoderm of the third pharyngeal pouch, and is subsequently colonized by T-cell precursors derived from hematopoietic stem cells in the aorta-gonad-mesonephros region. To explore this process we made use of the developmental and genetic advantages of the vertebrate zebrafish. To identify novel genes involved in early lymphopoiesis or thymopoiesis, mutations were generated in males using chemical mutagenesis. Eggs from female F1 progeny of ENU treated AB males and wildtype AB females were subjected to early pressure, and gynogenetic diploid embryos were grown to day 5. Embryos were then analyzed by whole mount in-situ hybridization (WISH) using the zebrafish orthologs of rag-1 and alpha embryonic globin probes. This screen allowed us to isolate several mutants of T-cell development. Of the 23 potential mutants that were found in the initial phase of our screen, eight recessive mutants with decreased or absent rag-1 and ***ikaros*** staining by WISH have been confirmed by incrossing of the F2 generation and fall into six complementation groups, all named for teas (for T cell and Thymic mutants). Four of these have been assigned to a linkage group by meiotic mapping. All of these mutants have normal erythropoiesis based on globin expression. A variable degree of arch abnormalities is found in four of the six groups. Neural crest cell migration and pharyngeal endoderm are normal in the mutants based on WISH. One of these mutants, earl grey, shows normal erythropoiesis and myelopoiesis, but complete absence of T lymphopoiesis, as well as abnormal arches and thymic development. We have undertaken a positional cloning approach and have defined flanking genetic markers within 0.4 cM (16/3440 meiotic recombinants) and 0.35 cM, respectively of the mutated gene. We have also established a ***PAC*** contig in the area of the earl grey gene. Fine mapping of the remaining mutants is ongoing. Cloning of the affected genes will help elucidate the steps involved in physiologic T cell and/or thymic epithelial development, and may also help identify genes affected in pathologic states, such as the DiGeorge syndrome.

L3 ANSWER 9 OF 9 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 3
AN 2000:61885 BIOSIS
DN PREV200000061885
TI Human Aiolos, an ***Ikaros*** -related zinc finger DNA binding protein: cDNA cloning, tissue expression pattern, and chromosomal mapping.
AU Hosokawa, Yoshitaka [Reprint author]; Maeda, Yumiko; Takahashi, Ei-ichi; Suzuki, Mikio; Seto, Masao
CS Laboratory of Chemotherapy, Aichi Cancer Center Research Institute, 1-1 Kanakoden, Chikusa-ku, Nagoya, 464-8681, Japan
SO Genomics, (Nov. 1, 1999) Vol. 61, No. 3, pp. 326-329. print.
CODEN: GNMCEP. ISSN: 0888-7543.
DT Article
LA English
ED Entered STN: 9 Feb 2000
Last Updated on STN: 3 Jan 2002
AB The ***Ikaros*** gene (symbol ZNFN1A1) encodes the hematopoietic zinc finger DNA binding protein, which is now recognized as a central regulator of lymphoid differentiation and has been implicated in leukemogenesis.

Recently, an ***Ikaros*** -related zinc finger protein, called Aiolos (ZNFN1A3), has been identified and characterized, thus establishing the presence of a gene family whose members may be hematopoietic transcription factors. Among Aiolos-mutant mice, development of B-cell lymphoma was frequently seen. As an initial approach to examining the possible involvement of Aiolos in the pathogenesis of human lymphoid proliferative disease, we isolated cDNA clones for human Aiolos from a B-cell cDNA library. The human Aiolos protein predicted from the cDNA sequence consists of 509 amino acid residues and shares 86% sequence identity with its mouse counterpart. As in the case with mouse Aiolos, no isoform for human Aiolos has been found. Northern blot analysis of various human tissues revealed that the Aiolos transcripts are expressed most strongly in peripheral blood leukocytes, the spleen, and the thymus, supporting the notion that Aiolos plays an important role in lymphoid lineages. Fluorescence in situ hybridization using a ***BAC*** clone established that the Aiolos gene is mapped to human chromosome band 17q11.2.

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L4 19 L1 AND OCT-1

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 9 DUP REM L4 (10 DUPLICATES REMOVED)

=> s l5 and Py<=2001
2 FILES SEARCHED...
L6 6 L5 AND PY<=2001

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 6 ANSWERS - CONTINUE? Y/(N):y

L6 ANSWER 1 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:223833 BIOSIS
DN PREV200200223833
TI Molecular cloning and chromosomal localization of the murine H-K-ATPase alpha2 subunit gene.
AU Kone, Bruce C. [Reprint author]; Kuncewicz, Teresa [Reprint author]
CS Division of Renal Diseases and Hypertension, University of Texas Medical School-Houston, Houston, TX, USA
SO Journal of the American Society of Nephrology, (September, 2000) Vol. 11, No. Program and Abstract Issue, pp. 31A. print.
Meeting Info.: 33rd Annual Meeting of the American Society of Nephrology and the 2000 Renal Week. Toronto, Ontario, Canada. October 10-16, 2000. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 3 Apr 2002
Last Updated on STN: 3 Apr 2002
AB The H-K-ATPase alpha2 (HKalpha2) subunit plays important roles in sodium, potassium and acid-base homeostasis. Mice genetically deficient in this gene exhibit fecal potassium wasting and profound hypokalemia during dietary potassium deprivation. The HKalpha2 gene is principally expressed in kidney and colon and is differentially regulated by aldosterone and potassium restriction in the two organs. Despite this evidence for complex regulatory controls, nothing is known of the genomic structure, organization or chromosomal localization of the HKalpha2 gene. To gain insights into structure-function aspects of the gene, we used an HKalpha2-specific cDNA probe to screen an 129/SvJ mouse genomic library by filter hybridization. A clone corresponding to 15 kB of the 5 prime-flanking sequence extending into intron 8 of the HKalpha2 gene was isolated. 2.6 kB of the proximal promoter region was sequenced and revealed multiple potential cis-elements, including a GRE and sites for CREB/ATF, AP-1, Sp1, ***Oct*** - ***1*** , NF-kappaB, C/EBP, and gut-enriched Kruppel-like factor. Fluorescence in situ hybridization analysis of normal metaphase chromosomes showed cohybridization of the HKalpha2 genomic probe with a probe specific for the centromeric region of chromosome 14, mapping the HKalpha2 gene to mouse chromosome band 14C3.
The entire HKalpha2 gene was then cloned from a murine ***BAC*** library using PCR generated HKalpha2-specific cDNA probes and filter hybridization. We have thus far sequenced the first 8 exons and mapped the intron/exon boundaries of this region. Since gene control elements may be positioned throughout the gene, this clone will allow detailed analysis of HKalpha2 gene transcription in vitro and in vivo. Moreover it will allow direct structural comparison to the putative HKalpha2 ortholog, human ATPIAL1.

L6 ANSWER 2 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2000:346542 BIOSIS
DN PREV200000346542
TI Isolation and characterization of the human UGT2B7 gene.
AU Carrier, Jean-Sebastien; Turgeon, David; Journault, Kim; Hum, Dean W.; Belanger, Alain [Reprint author]
CS Laboratory of Molecular Endocrinology, CHUL Research Center, 2705 Laurier Blvd., Quebec, G1V 4G2, Canada
SO Biochemical and Biophysical Research Communications, (June 7, 2000) Vol. 272, No. 2, pp. 616-621. print.
CODEN: BBRCA9. ISSN: 0006-291X.
DT Article
LA English
ED Entered STN: 16 Aug 2000
Last Updated on STN: 7 Jan 2002
AB Glucuronidation is a major pathway involved in the metabolism of drugs and numerous endogenous compounds, such as bile acids and steroid hormones. The enzymes responsible for this conjugation reaction are UDP-glucuronosyltransferases (UGT). Among the UGT2B subfamily, UGT2B7, a
UGT enzyme present in the liver and several steroid target tissues, is an important member since it conjugates a large variety of compounds including estrogens, androgens, morphine, AZT, and retinoic acid. Although this enzyme is well characterized, the gene encoding the UGT2B7 protein and its promoter region remain unknown. In this article, we report the genomic organization and the promoter region of the human UGT2B7 gene. To isolate this gene, a P-1 ***artificial*** ***chromosome*** (***PAC***) library was screened with a full length UGT2B7 probe and a clone of approximately 100 kb in length was isolated. In addition to the UGT2B7 gene, this ***PAC*** contains two other UGT2B genes previously characterized, namely UGT2B26P and UGT2B27P. The
UGT2B7 gene is composed of six exons spanning approximately 16 kb, with introns ranging from 0.7 to 4.2 kb. The 5'-flanking region of the human UGT2B7 gene contains several potential cis-acting elements such as ***Oct*** - ***1*** , Pbx-1, and C/EBP. Only one TATA-box at nucleotide - 106 was found within the first 500 nucleotides relative to the adenine base of the initiator ATG codon. Characterization of the UGT2B7 gene provides insight into the organization and regulation of this important metabolic gene.

L6 ANSWER 3 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2000:113766 BIOSIS
DN PREV200000113766
TI Isolation and characterization of the human UGT2B15 gene, localized within a cluster of UGT2B genes and pseudogenes on chromosome 4.
AU Turgeon, David; Carrier, Jean-Sebastien; Levesque, Eric; Beatty, Barbara G.; Belanger, Alain [Reprint author]; Hum, Dean W. [Reprint author]
CS Laboratory of Molecular Endocrinology, Laval University, Laval, Quebec, G1V 4G2, Canada
SO Journal of Molecular Biology, (Jan. 21, 2000) Vol. 295, No. 3, pp. 489-504. print.
CODEN: JMOBAK. ISSN: 0022-2836.
DT Article
LA English
ED Entered STN: 29 Mar 2000
Last Updated on STN: 3 Jan 2002
AB Glucuronidation is a major pathway of androgen metabolism and is catalyzed by UDP-glucuronosyltransferase (UGT) enzymes. UGT2B15 and UGT2B17 are 95%
identical in primary structure, and are expressed in steroid target tissues where they conjugate C19 steroids. Despite the similarities, their regulation of expression are different; however, the promoter region and genomic structure of only the UGT2B17 gene have been characterizedX to date. To isolate the UGT2B15 gene and other novel steroid-conjugating UGT2B genes, eight P-1-derived ***artificial*** ***chromosomes*** (***PAC***) clones varying in length from 30 kb to 165 kb were isolated. The entire UGT2B15 gene was isolated and characterized from the ***PAC*** clone 21598 of 165 kb. The UGT2B15 and UGT2B17 genes are highly conserved, are both composed of six exons spanning approximately 25 kb, have identical exon sizes and have identical exon-intron boundaries. The homology between the two genes extend into the 5'-flanking region, and contain several conserved putative cis-acting elements including Pbx-1, C/EBP, AP-1, ***Oct*** - ***1*** and NF/kappaB. However, transfection studies revealed differences in basal promoter activity between the two genes, which correspond to regions containing non-conserved potential elements. The high degree of homology in the 5'-flanking region between the two genes is lost upstream of -1662 in UGT2B15, and suggests a site of genetic recombination involved in duplication of UGT2B genes. Fluorescence in situ hybridization mapped the UGT2B15 gene to chromosome 4q13.3-21.1. The other ***PAC*** clones isolated contain exons from the UGT2B4, UGT2B11 and UGT2B17 genes. Five novel exons, which are highly homologous to the exon 1 of known UGT2B genes, were also identified; however, these exons contain premature stop codons and represent the first recognized pseudogenes of the UGT2B family. The localization of highly homologous UGT2B genes and pseudogenes as a cluster on chromosome 4q13 reveals the complex nature of this gene locus, and other novel homologous UGT2B genes encoding steroid conjugating enzymes are likely to be found in this region of the genome.

L6 ANSWER 4 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:361904 BIOSIS
DN PREV199800361904
TI Genomic organization and chromosomal localization of the murine epididymal retinoic acid-binding protein (mE-RABP) gene.
AU Lareyre, Jean-Jacques; Mattei, Marie-Genevieve; Kasper, Susan; Ong, David E.; Matusik, Robert J.; Orgebin-Crist, Marie-Claire [Reprint author]
CS Cent. Reproductive Biol. Res., Vanderbilt Univ., Sch. Med., Medical Center N., Room D2303, Nashville, TN 37233-2633, USA
SO Molecular Reproduction and Development, (Aug., 1998) Vol. 50, No. 4, pp. 387-395. print.
CODEN: MREDEE. ISSN: 1040-452X.

DT Article
LA English
ED Entered STN: 27 Aug 1998
Last Updated on STN: 27 Aug 1998
AB The murine epididymal retinoic acid-binding protein (mE-RABP) is specifically synthesized in the mouse mid/distal caput epididymidis and secreted in the lumen. In this report, we have demonstrated by Southern blot analysis of genomic DNA that mE-RABP is encoded by a single-copy gene. A mouse 129/SvJ genomic bacteria) ***artificial*** ***chromosome*** (***BAC***) library was screened using a cDNA encoding the minor form of mE-RABP. One positive ***BAC*** clone was characterized and sequenced to determine the nucleotide sequence of the entire mE-RABP gene. The molecular cloning of the mE-RABP gene completes the characterization of the 20.5-kDa-predicted preprotein leading to the minor and major forms of ME-RABP. Comparison of the DNA sequence of the promoter and coding regions with that of the rat epididymal secretory protein I (ESP 1) gene showed that the mE-RABP gene is the orthologue of the ESPI gene that encodes a rat epididymal retinoic acid-binding, protein. Several regulatory elements, including a putative androgen receptor binding site, "CACCC-boxes," NF-1, ***Oct*** - ***1*** , and SP-1 recognition sites, are conserved in the proximal promoter. Analysis of the nucleotde sequence of the mE-RABP gene-revealed the presence of seven exons and showed that the genomic organization is highly related to other genes encoding lipocalins. The mE-RABP gene was mapped by fluorescent in situ hybridization to the (A3-B) region of the murine chromosome 2. Our data, combined with that of others, suggest that the proximal segment of the mouse chromosome 2 may be a rich region for genes encoding lipocalins with a genomic organization highly related to the mE-RABP gene.

L6 ANSWER 5 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1995:171277 BIOSIS
DN PREV199598185577
TI The gene encoding the VP16-accessory protein HCF (HCFC1) resides in human
Xq28 and is highly expressed in fetal tissues and the adult kidney.
AU Wilson, Angus C.; Parrish, Julia E.; Massa, Hillary F.; Nelson, David L.; Trask, Barbara J.; Herr, Winship [Reprint author]
CS Cold Spring Harbor Lab., P.O. Box 100, Cold Spring Harbor, NY 11724, USA
SO Genomics, (1995) Vol. 25, No. 2, pp. 462-468.
CODEN: GNMCEP. ISSN: 0888-7543.
DT Article
LA English
ED Entered STN: 26 Apr 1995
Last Updated on STN: 26 Apr 1995
AB After herpes simplex virus (HSV) infection, the viral regulatory protein VP16 activates transcription of the HSV immediate-early promoters by directing complex formation with two cellular proteins, the POU-homeodomain transcription factor ***Oct*** - ***1*** and the host cell factor HCF. The function of HCF in uninfected cells is unknown. Here we show by fluorescence in situ hybridization and somatic cell hybrid analysis that the gene encoding human HCF, HCFC1, maps to the q28 region of the X chromosome. Yeast ***artificial*** ***chromosome*** and cosmid mapping localizes the HCFC1 gene within 100 kb distal of the renal vasopressin type-2 receptor (V2R) gene and adjacent to the renin-binding protein gene (RENBP). The HCFC1 gene is apparently unique. HCF transcripts and protein are most abundant in fetal and placental tissues and cell lines, suggesting a role in cell proliferation. In adults, HCF protein is abundant in the kidney, but not in the brain, a site of latent HSV infection and where HCF levels may influence progression of HSV infection.

L6 ANSWER 6 OF 6 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1993:319912 BIOSIS
DN PREV199396028262
TI Chromosomal structure and expression of the human OTF1 locus encoding the ***Oct*** - ***1*** protein.
AU Sturm, Richard A. [Reprint author]; Cassady, Jennifer L.; Das, Gokul; Romo, Anthony; Evans, Glen A.
CS Cent. Mol. Biol. and Biotechnol., Univ. Queensland, Qld 4072, Australia
SO Genomics, (1993) Vol. 16, No. 2, pp. 333-341.
CODEN: GNMCEP. ISSN: 0888-7543.
DT Article
LA English
ED Entered STN: 12 Jul 1993
Last Updated on STN: 12 Jul 1993
AB The genomic structure of the POU domain containing ***oct*** - ***1*** gene (OTF1 locus) coding region has been determined using human DNA recombinant bacteriophage and a yeast ***artificial*** ***chromosome*** clone. The gene is encoded by 16 exons spanning over 150 kb, and the ***Oct*** - ***1*** protein reading frame has been extended to 766 amino acids. The exonic structure has been compared to the mouse Oct-2 protein and reveals a conservation of exon-intron boundaries as well as protein sequence similarity. To provide insight into ***Oct*** - ***1*** control of transcriptional regulation during the cell-cycle the expression of the ***oct*** - ***1*** gene was examined during cellular DNA replication and shows that the steady-state level of the ***oct*** - ***1*** mRNA is not S-phase regulated.

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FULL ESTIMATED COST		61.32	61.53

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CA SUBSCRIBER PRICE		-3.47	-3.47

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